

The effect of acute alcohol intoxication on social inhibition and theory of mind:
A social lubricant or social depressant?

Emma Grace Johnson

A report submitted as a partial requirement for the degree of Bachelor of Arts with
Honours in Psychology at the University of Tasmania, 13 October 2016.

Statement of Sources

I declare that this report is my own original work and that contributions of others have been duly acknowledged.

Emma Johnson

/ /
Date

Acknowledgements

Thanks must go to Tasmania Police for the loan of the breathalyser that made this study possible. Special thanks are extended to Senior Sergeant Nick Clark and Constable Sharon Murphy for their assistance throughout the study, with re-calibration and the provision of an emergency breathalyser.

Thank you to all the people who volunteered their time for this study.

To my supervisor, Dr Cynthia Honan, thank you for all you have done for me this year. Thank you, especially, for your faith in me, your encouragement, counsel and advice. You have been a wonderful supervisor.

To Sarah, I couldn't have asked for a better colleague to work on this project with! Thank you for your organisation, professionalism, and your friendship.

To Uncle David, thank you for proof reading, and for being so kind with your feedback.

Thank you to my brothers: Andrew, for walking me through pharmacokinetics, spread sheets, maths and physics, and for getting drunk for me all the way over in Chicago just to ensure the equation worked! To Paul, thank you for participating and all the caffeine and support!

Table of Contents

List of Figures and Tables.....	v
List of Acronyms	vi
Abstract	1
Social Cognition.....	4
Theory of Mind	5
The Alcohol Myopia Model.....	6
Alcohol and Inhibitory Control.....	8
The Current Study	11
Hypotheses	12
Method	13
Participants.....	13
Materials.....	15
Eligibility Assessments	15
Baseline Measures.....	16
Alcohol Intoxication Measures	17
Experimental Measures	18
Procedure.....	20
Design	22
Statistical Analyses	22
Results	24
Eligibility and Baseline Measures.....	24
Alcohol Intoxication Measures	25
Experimental Measures	27
Discussion	34
References	41
Appendix A Ethics Approval	52
Appendix B Noticeboard Advertisement	54
Appendix C Information Sheet	55
Appendix D Consent Form	59
Appendix E Widmark Equation	62
Appendix F Social Disinhibition Task Sample Items	63

List of Figures and Tables

Table 1 <i>Comparisons of Demographic Information Between Groups.</i>	14
Table 2 <i>Comparisons of Group Characteristics on Eligibility Measures.</i>	25
Figure 1. Subjective ratings of effects of alcohol on sedation and stimulation for each group at each time point.....	26
Figure 2. Rate of error on the Flanker task by compatibility. Error bars represent standard error values.	28
Figure 3. Mean reaction time as a function of compatibility and treatment group. Error bars represent standard errors.	29
Figure 4. Responses to Item Type and each Part stratified by condition.....	31
Table 3 <i>Correlations Between SDT Score For Item Type (Parts A & B) and the Go/No-Go and Flanker Tasks</i>	33

List of Acronyms

AUDIT	Alcohol Use Disorders Identification Test
BAES	Biphasic Alcohol Effects Scale
BMI	Body Mass Index
BrAC	Breath Alcohol Concentration
BRS	Beverage Rating Scale
FIML	Full Information Maximum Likelihood
K10	Kessler Psychological Distress Scale
SDT	Social Disinhibition Task
TBI	Traumatic Brain Injury
TLFB	Timeline Followback

The effect of acute alcohol intoxication on social inhibition and theory of mind:

A social lubricant or social depressant?

Word Count: 9984

Emma Grace Johnson

Abstract

Alcohol intoxication is associated with socially disinhibited behaviours that may reflect impaired social cognitive abilities that guide social behaviour. The effects of alcohol on social cognition, and how this may contribute to disinhibited behaviour is relatively poorly studied. The aims of this study were to examine whether intoxicated individuals could inhibit negative responses to negative social information, whether these difficulties were reliant on theory of mind ability, and whether intoxicated individuals were able to adjust verbal responses when provided with guidelines about how to respond. Sixty-four males and females aged between 18 and 34 were recruited from the University of Tasmania and wider community to participate in the study. Participants consumed a beverage containing either alcohol or placebo before completing a Flanker task, a Go/No-Go task, and a newly developed measure of social disinhibition, the Social Disinhibition Task. Results indicate that alcohol intoxicated individuals can inhibit negative responses to negative social information, but display difficulty inhibiting negative responses to social information requiring ToM ability. Intoxicated individuals were able to adjust responses when required. These findings extend current understandings of the mechanisms involved in negative social behaviours following acute alcohol administration and may have important implications for development of public policy.

The effect of acute alcohol intoxication on social inhibition and theory of mind:

A social lubricant or social depressant?

The consumption of alcohol is ubiquitous to Australian social settings, and is synonymous with recreation, entertainment and relaxation. It is well established that alcohol produces a broad range of noticeable changes to cognition and behaviour which, in turn, affect demeanour and social conduct (Fillmore, Vogel-Sprott, & Gavrilescu, 1999; Heath & Hardy-Vallée, 2015). These changes are often manifested as socially disinhibited behaviour. Social disinhibition may be defined as a diminished concern for self-presentation and evaluative pressure in a social context, exhibited as impulsive or unrestrained behaviour (Freeman, Friedman, Bartholow, & Wulfert, 2010). The disinhibiting properties of alcohol are often viewed as a positive effect, evidenced by the frequent use of the drug as a social lubricant (Sayette et al., 2012) to increase self-confidence and self-disclosure (Monahan & Lannutti, 2000), facilitate conversation (Grace, Moore, & Northcote, 2009; Sayette et al., 2012), and reduce anxiety (Monahan & Lannutti, 2000). Indeed, alcohol is generally consumed for its anxiolytic properties in order to modulate affective states by inducing pleasant, relaxing effects, and attenuating negative mood states, stress and tension (Hull & Slone, 2004; Lithari et al., 2012; Padula et al., 2011; Sripada, Angstadt, McNamara, King, & Phan, 2011; Vengeliene, Bilbao, Molander, & Spanagel, 2008).

However, alcohol consumption does not consistently result in positive social behaviours from one drinking episode to the next (Attwood & Munafò, 2014; Giancola, Josephs, Parrott, & Duke, 2010; Steele & Josephs, 1990). This is exemplified by the variety of behaviours exhibited by any given individual following acute alcohol consumption, which may range, depending on the situation, from increased sociability to highly antisocial conduct (Giancola et al., 2010; Steele &

Josephs, 1990). In reality, despite consuming alcohol for its pleasurable effects, intoxicated individuals frequently exhibit socially inappropriate behaviours that contribute to negative outcomes such as poor decision making, sexual risk taking and increased aggression (Assaad et al., 2006; Lopez-Caneda, Rodriguez Holguin, Cadaveira, Corral, & Doallo, 2014; Morgan & McAtamney, 2009; Ostling & Fillmore, 2010). This is because social disinhibition may also be manifested as a lack of restraint on verbal, physical or sexual behaviours that are inappropriate to social or cultural expectations and norms (Arciniegas & Wortzel, 2014; Osborne-Crowley & McDonald, 2016).

Socially disinhibited behaviours associated with acute alcohol intoxication may impose enormous direct and indirect costs and harms to individuals and the community (Manning, Smith, & Mazerolle, 2013). For example, alcohol consumption is a common factor in violent crimes, including domestic abuse and sexual aggression (Manning et al., 2013). A high proportion of assaults involve intoxicated individuals as either offenders or victims (Morgan & McAtamney, 2009), and alcohol was implicated in 47% of all homicides in Australia between 2000 and 2006 (Dearden & Payne, 2009). The latest available data from a national Australian survey indicated that 86% of respondents believed drunken and disorderly behaviour to be a problem in their neighbourhood (Steering Committee for the Review of Commonwealth/State Service Provision, 2009). The implementation of “lock-out laws” in Sydney’s central business district is a recent indicator of the prevalence of alcohol over-consumption in social settings and the antisocial behavioural consequences of that consumption that affect the wider community.

Despite a large volume of research examining the effects of alcohol on behaviour, the mechanisms by which intoxicated individuals engage in socially

disinhibited behaviours are not fully understood (Attwood & Munafò, 2014). To date, research has established that alcohol directly impacts on neural systems involving the prefrontal regions of the brain, impairing cognitive processes that are necessary for regulation of behaviour (Attwood & Munafò, 2014; Pedersen, Vasquez, Bartholow, Grosvenor, & Truong, 2014). However, alcohol expectancies and individual differences aside, the pharmacological effects of the drug do not explain the inconsistent behavioural manifestations exhibited by intoxicated individuals (Heath & Hardy-Vallée, 2015; Steele & Josephs, 1990). Two main approaches have been applied to explain how alcohol intoxication contributes to disinhibited behaviours. Cognitive neuroscience tends to apply a bottom-up experimental approach focused on investigating the effects of alcohol on inhibitory mechanisms of behavioural control (Fillmore & Weafer, 2004). In comparison, a top-down, theoretical approach typically referred to as the Alcohol Myopia Model (AMM) focuses on the social and situational determinants of the effects of alcohol on behaviour (Giancola et al., 2010; Steele & Josephs, 1990). However, these models of alcohol related behaviour, discussed in more detail later, do not take into account the important role of social cognition in guiding social behaviour.

Social Cognition

Social cognition encompasses processes related to empathy, recognition of emotional states of others, understanding the perspectives of others and gauging the intentions behind the actions of others (theory of mind), and inhibiting automatic responses in favour of more socially acceptable behaviours (McDonald, Honan, Kelly, Byom, & Rushby, 2013). Social cognition therefore regulates how a person perceives, understands, interprets, and responds to their social environment. The ability to process social signals within the environmental context is therefore

necessary to guiding social behaviours (Attwood & Munafò, 2014). As alcohol alters social behaviour, and social cognition guides social behaviour, it is likely that socially disinhibited behaviours exhibited during episodes of intoxication reflect interference with social cognitive processes (Dolder et al., 2016). Whilst a number of studies have examined the effect of alcohol on emotion perception (see Attwood and Munafò, 2014; Kamboj et al., 2013; Dolder et al., 2016) there is a paucity of literature examining other important aspects of social cognition such as theory of mind (ToM).

De Wit and Dickinson's (2009) Associative Theory of Goal Directed Behaviour indicates it is reasonable to expect that effective suppression (inhibition) of an inappropriate behaviour requires immediate responsiveness to environmental, physiological and emotional events. For example, in order to inhibit a response, an individual must first process situational and contextual variables, and identify whether the automatic response is appropriate. This ability of an individual to accurately evaluate the behaviours and thoughts of others, determine the appropriateness of their own response, and adjust that response to be more socially appropriate, is an indicator of successful adjustment to social norms and expectations. This requires intact higher order social cognition abilities, especially theory of mind (ToM).

Theory of Mind

Theory of mind (ToM) refers to perspective taking, the ability to perceive social cues and use them to make inferences about the mental states of other people in order to evaluate and predict their social behaviours (Bibby & McDonald, 2005). These higher order evaluations are fundamental to informing 'appropriate' social behaviours. It is possible that alcohol specific socially disinhibited behaviours are

mediated by both impaired basic inhibitory mechanisms that are thought to suppress inappropriate behaviour, and impaired ToM ability.

Only one known study has investigated the effects of acute alcohol on ToM ability (Mitchell, Beck, Boyal, & Edwards, 2011). In this study, intoxicated individuals were impaired on two measures of ToM ability, the *The Faux Pas Recognition Test* (Stone, Baron-Cohen, & Knight, 1998) which involves reading lengthy verbal vignettes to participants, and the *Reading the Mind in the Eyes Test* (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001) which involves looking at still black and white photographs of the eyes only and selecting from a list of response options what the person is thinking or feeling.

The Alcohol Myopia Model

The dominant theory to explain intoxicated behaviours is the alcohol myopia model (AMM) (Steele & Josephs, 1990). This model suggests that alcohol impairs cognitive processes necessary for perception, attention and subsequent information processing of external and internal cues (Giancola et al., 2010). With fewer attentional resources available, intoxicated individuals attend to fewer environmental cues. Consequently, only salient information is processed, and subtle information, (referred to hereon as inhibitory cues) that may be vital to correct interpretation of the situation is overlooked, such as consideration of consequences or detecting sarcasm (Denson et al., 2008; Pedersen et al., 2014; Steele & Josephs, 1990). AMM posits that inhibitory cues require greater cognitive resources to process information, as would be the case when needing to detect the particular social norms that are applied to a more novel social context (e.g., black tie event) (Denson et al., 2008).

To provide an example of a typical AMM study design, Denson et al. (2008) examined the effects of high and low aggressive-cue salience on acts of displaced

aggression in an intoxicated sample. They found that intoxicated individuals were more likely to engage in aggressive behaviour following high relative to low salience aggression-inducing cues. In this context, the inhibitory cues that would normally prevent a person from aggression, such as consideration of appropriate social conduct and potential consequences, were less salient and required information processing abilities that had been disrupted by alcohol intake (Denson et al., 2008).

Because of its simplicity, the AMM paradigm possesses great versatility of application across all manifestations of intoxicated behaviour. AMM has therefore been used to account for a broad range of intoxicated behaviours, including aggression, negative affect, and self-awareness (Giancola et al., 2010; Monahan & Lannutti, 2000). It has also been applied to the social behaviour of rats following acute alcohol administration (Grant & Macdonald, 2005) and even to situations where alcohol has not even been administered. For example, Gable, Mechin, and Neal (2016) demonstrated what they referred to as ‘virtual myopia’ following exposure to alcohol-related cues without the presence of intoxication. The authors suggested that similar attentional narrowing as described in AMM occurred after participants were merely presented with pictures of alcoholic beverages.

As a theoretical framework, AMM offers a persuasive explanation for behaviour following acute alcohol consumption. However, AMM does not satisfactorily describe the specific cognitive mechanisms disrupted by alcohol. AMM broadly implies that attention is the primary cognitive process disrupted by alcohol that causes disinhibited behaviour, yet research indicates that other cognitive processes are also impaired by alcohol, response inhibition in particular (Abroms, Fillmore, & Marciszynski, 2003; Lyvers & Tobias-Webb, 2010). Additionally, successful social behaviour relies not just on perception of salient and subtle social

cues, but also on successful interpretation of contextual social information. For example, successful banter between friends requires the ability of both parties to process multiple social stimuli in order to respond appropriately. Both dialogue and facial expressions serve as cues of varying salience that imply sarcasm or a joke rather than a threat, yet it is the context of the friendship that determines that there is no threat and that aggression in this situation would be inappropriate. Social knowledge and higher order social cognitive abilities such as emotion processing and ToM are therefore essential to correct interpretation of social contexts, and in reference to AMM, interpreting relevant inhibitory cues.

Alcohol and Inhibitory Control

In contrast with AMM which describes alcohol as impairing perception, attention and information processing, cognitive studies have identified inhibitory control mechanisms as being particularly sensitive to the effects of alcohol (Abroms et al., 2003; Ostling & Fillmore, 2010). Inhibitory control is a core functions of the frontal-subcortical executive system, and is necessary to behaviour because of the role it plays in enabling adaptation and regulation of behaviour according to environmental demands (Dimoska-Di Marco, McDonald, Kelly, Tate, & Johnstone, 2011; Lopez-Caneda et al., 2014). As described by Dimoska-Di Marco et al. (2011), inhibitory control involves the capacity to interrupt or delay an activated response or course of action and provide a more appropriate controlled response while suppressing the automatic response. Response inhibition is a component of inhibitory control which refers specifically to the ability to the suppression of an automatic response when required (Burden et al., 2010; Diamond, 2013). Response inhibition is typically assessed using tasks that require either inhibition of an automatic response in favour of a task-relevant response (such as the Go/No-Go

[GNG] or Stop Signal [SS] tasks). Inhibitory control, on the other hand, is assessed using tasks such as Flanker or Stroop tasks which require deliberate perceptual and attentional inhibition of distracting stimuli (Goghari & MacDonald, 2009; Wager et al., 2005; Weafer & Fillmore, 2016).

Studies utilising these tasks provide evidence of alcohol related impairment to the ability to inhibit behaviour (e.g., Fillmore & Vogel-Sprott, 2000; Abroms et al., 2003; de Wit, Crean & Richards, 2000; Marczinski, Abroms, Van Selst, & Fillmore, 2005; Mulvihill, Skilling, & Vogel-Sprott, 1997). Impairment associated with these cognitive mechanisms is thus believed to contribute to the display of impulsive, aggressive, and other socially inappropriate behaviours typically observed in intoxicated individuals (Abroms et al., 2003; Chambers, Garavan, & Bellgrove, 2009; Fillmore et al., 1999; Giancola, 2007; Marczinski et al., 2005; Pedersen et al., 2014). A number of studies have demonstrated that even a low dose of alcohol reduces the ability to inhibit prepotent (i.e. automatic or habitual) responses (see de Wit et al., 2000; Marczinski et al., 2005; Montgomery, Ashmore, & Jansari, 2011; Weafer & Fillmore, 2012; Weafer & Fillmore, 2016).

Weafer and Fillmore (2012) investigated the effects of acute alcohol on measures of both behavioural response inhibition and attentional inhibition in 48 participants (27 men and 21 women). Participants completed a cued GNG task as a measure of behavioural response inhibition, and a delayed ocular return task measuring attentional inhibition or control across three conditions (placebo, .45 g/kg alcohol and .65 g/kg alcohol). Impairment of behavioural response inhibition (evidenced by increased failures to inhibit responses to no-go targets [errors of commission] and by slowed reaction time to go targets) and attentional inhibition (evidenced by increased premature saccades) was demonstrated in both alcohol

conditions. Correlational analyses did not indicate any association between behavioural response and attentional inhibition tasks. Weafer and Fillmore concluded that whilst both tasks are equally sensitive to the effects of alcohol, they represent distinct mechanisms of inhibitory control behaviours.

In a similar within groups placebo balanced design, Abroms et al. (2003) measured response inhibition performance of 29 men on a cued reaction time task before and after a 0.65 g/kg dose of alcohol. Their results indicated that alcohol impaired the ability to suppress a prepotent response. Likewise, Marcziński and Fillmore (2005) found that intoxicated individuals were impaired in their performance on a GNG task following both 0.45 g/kg, and 0.65g/kg doses of alcohol. Specifically, alcohol impaired both suppression and activation of a response on incongruent trials, but not on congruent trials.

In a separate non-alcohol study of 71 Australian University students from non-Asian background, von Hippel and Gonsalkorale (2005) investigated the effects of interrupted inhibitory control in a social context, to examine the link between inhibitory control and socially inappropriate behaviour. Participants completed baseline assessments of a Stroop task before participating in either a high or low social pressure to give a socially acceptable response to an offer of an unappealing food (a chicken foot). The high social pressure group were offered the chicken foot by an Asian experimenter, who described it as a valued cultural dish. The low social pressure group were offered the chicken foot by a Caucasian experimenter, with the same description of the chicken foot. Performance on the Stroop task was found to predict the degree to which negative responses were inhibited, i.e., those who had poor inhibitory ability were more likely to make a socially inappropriate response. These findings indicate that inhibitory control may contribute to regulation of social

responses. However, no known studies have integrated both inhibitory control and social context (i.e. where social cognition abilities are also required) in studies of acute alcohol administration to examine how intoxicated individuals might inhibit their inappropriate responding to negative social information. Developing an understanding of how these processes interact may be vital to furthering our understanding of the mechanisms involved in negative social behaviours following acute alcohol administration.

The Current Study

This study will first attempt to replicate a prior finding that alcohol impairs ToM (Mitchell et al., 2011) and extend on the current literature by examining whether alcohol intoxicated individuals are impaired at inhibiting negative automatic responses. This will be investigated by using a newly developed measure of social disinhibition that assesses the ability to inhibit automatic negative social utterances in favour of more socially appropriate responses, when presented with negative social information (Honan, Allen, Fisher, Osborne-Crowley & McDonald, in press). This negative social information will include both salient negativity (e.g., a message that clearly portrays a negative social scene), and faux pas items where negativity is less salient and requires higher level ToM ability for it to be interpreted. Examination of the interaction between inhibition ability and the two types of social information will provide an indication of whether ToM ability facilitates social inhibition ability. A supplemental aim is to investigate whether intoxicated individuals are able to adjust their responses when provided with guidelines about how to respond. A placebo balanced design will be used to control for alcohol expectancies.

Hypotheses

To examine the effects of acute alcohol intoxication on social disinhibition, theory of mind ability, and the ability for intoxicated individuals to adjust verbal responses according to specific guidelines, the following hypotheses are proposed:

(1) Consistent with prior research demonstrating alcohol induced impairments to basic inhibitory control mechanisms (Abroms et al., 2003; Marczinski & Fillmore, 2005; Weafer & Fillmore, 2012), alcohol intoxicated individuals will perform more poorly on laboratory based inhibitory control tasks (Flanker) and response inhibition (cued Go/No-Go) tasks than individuals administered a placebo. (2) In the social disinhibition task, alcohol intoxicated individuals will display more difficulties in inhibiting automatic negative responses to negative social information than individuals administered a placebo. (3) Based on AMM, intoxicated individuals will be less able to detect negativity portrayed in the ToM/faux pas items (i.e., because the faux pas will be less salient to them due to expected ToM impairments).

Therefore, inhibition of automatic negative responses to ToM items will be less pronounced for intoxicated individuals than individuals administered a placebo. (4) As AMM suggests that intoxicated individuals attend only to salient cues (Denson et al., 2008; Steele & Josephs, 1990), and because alcohol impairs inhibitory control ability (Abroms et al., 2003; Marczinski & Fillmore, 2005; Weafer & Fillmore, 2012), it is expected that intoxicated individuals will be less able to inhibit their responses to salient negative social information to produce more socially acceptable, positive responses to negative social information when specifically asked to do so.

Method

Participants

An a priori power analysis (using G*Power 3.1.9.2) indicated that a sample of 46 participants (23 in each condition) would allow reliable (power = 0.81) detection of a statistically significant ($\alpha = .05$) effect, based on the estimated large effect size (Cohen's $d = .80$).

Participants were recruited from the University of Tasmania (UTAS) and the wider community through advertisement on noticeboard, social media, and the UTAS Discipline of Psychology webpage (Appendix A), and through verbal discussion in first year Psychology practical classes. Participants were quasi-randomly allocated (random assignment determined using the randomisation feature in Microsoft Excel) to either the alcohol or placebo condition to ensure an equally representative number of males and females in each group. Due to non-compliance to pre-study requirements (specifically, consumption of food or caffeine prior to the experimental session), non-attendance, and the withdrawal of one participant, the final sample consisted of 64 participants (32 males and 32 females) aged between 18 and 34 years. Thirty-one participants (16 males, 15 females) were assigned to the alcohol condition, and 33 participants (16 males, 17 females) to the placebo condition. Condition groups did not differ in age and years of education. Table 1 displays participants' descriptive information stratified by condition and inferential statistical information.

Table 1

Comparisons of Demographic Information Between Groups.

	Placebo		Alcohol		<i>t</i>	<i>df</i>	Sig	95% CI's	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>				Lower	Upper
Age [#]	22.70	4.80	24.55	3.38	1.78	54	.081	-.24	3.94
Gender	.52	.51	.48	.51	-.24	62	.806	-.29	.22
Education	11.73	.63	11.81	.87	.42	62	.677	-.30	.46

Note: [#]Equal variances not assumed statistic reported.

Strict eligibility requirements were implemented to recruit participants who were: (1) aged between 18-35 years; (2) within a body mass index (BMI) range of 18.5–29.9 (this criterion was included as BMI significantly influences the volume of distribution of alcohol (Maudens et al., 2014), and the equation used to calculate alcohol dosage (Dry, Burns, Nettelbeck, Farquharson, & White, 2012) is applicable only to underweight or normal weight persons); (3) moderate and regular consumers of alcohol, determined by self-reported consumption of at least two standard drinks on a single occasion within the previous fortnight on the Timeline Followback (Sobell & Sobell, 1992) and scores on the AUDIT (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001); (4) were fluent in written and spoken English; (5) had completed Year 10 or equivalent; and (6) had normal or corrected to normal vision. Exclusion criteria included: (1) regular tobacco use; (2) illicit drug use within the last month; (3) current use of any prescription medication (other than contraceptives); (4) history of any neurological condition (e.g., epilepsy, stroke); (5) current diagnosis of psychiatric disorder, or a score of 30 or above on the Kessler Psychological Distress

Scale (Kessler et al., 2002); and (7) a history of alcohol abuse evident via a score of 16 or higher on the Alcohol Use Disorders Identification Test (AUDIT; Babor et al., 2001).

Materials

Eligibility Assessments

Kessler Psychological Distress Scale (Kessler et al., 2002). The K10 is a 10-item self-report measure of feelings of distress experienced in the previous 30 days (e.g. “During the last 30 days, about how often did you feel tired out for no good reason?”). Participants indicate the extent to which they had these feelings on a 5-point Likert type scale, with response options ranging from 1 = ‘None of the time’ to 5 = ‘All of the time’. Ratings are summed to give a total ‘psychological distress’ score, with a maximum value of 50. Scores greater than 30 are indicative of higher levels of distress. The K10 is a well validated and reliable measure of psychological distress, and has been shown to have good psychometric properties to identify psychological distress in individuals with Alcohol Related Disorders, Cronbach’s $\alpha = .84$, sensitivity = .95 and specificity = .54 (Arnaud et al., 2010).

Alcohol Use Disorders Identification Test (AUDIT) Self-Report Version (Babor et al., 2001). A screening tool developed by the World Health Organisation (WHO) to identify alcohol or drug abuse, dependence disorder or use of alcohol at hazardous or harmful levels, the AUDIT consists of 10 items relating to the participant’s use of alcohol (e.g. “How often during the last year have you failed to do what was normally expected of you because of alcohol?”). Items are scored on a scale ranging from 0 to 4. Items are summed to obtain the total score. Total scores range from 0 to 40, with scores above 8 considered to be indicative of hazardous alcohol use, and scores over 16 indicative of a high level of alcohol problems. The AUDIT has good

psychometric properties. Cronbach's α coefficients have been reported between .83 and .94. The AUDIT also has high predictive validity, with sensitivity indices of .89 for abuse and .93 for dependence, and specificity indices of .93 for abuse and .95 for dependence (Meneses-Gaya et al., 2010).

Timeline Follow-Back Questionnaire (Sobell & Sobell, 1992). The TLFB is a measure of habitual drinking based on self-reported daily alcohol consumption patterns over the previous month. Participants are asked to indicate on a calendar the number of standard drinks they consumed on each day in the previous month. This was administered to obtain sample characteristic information and screen for drinking behaviour (i.e., participants must have had a minimum of 2 standard drinks on one occasion in the past fortnight to participate, and must not have consumed alcohol in the previous 24 hours). Good internal consistency, Cronbach's $\alpha = .84$ (Wennberg & Bohman, 1998) has been calculated for the TLFB, and it has been shown to have good retest reliability, convergent and discriminant validity with other measures (Fals-Stewart, O'Farrell, Freitas, McFarlin, & Rutigliano, 2000).

Baseline Measures

Frontal Assessment Battery: Inhibition Subtests (Dubois, Slachevsky, Litvan, & Pillon, 2000). These FAB subtests assess basic inhibitory control. Inter-rater reliability of the measure is good ($\kappa = .87$) and it has been validated for use in various population groups to identify frontal lobe dysfunction (Dubois et al., 2000). *Subtest 4, Conflicting Instructions*: Subjects provide an opposite response to the examiner's alternating signal, e.g. tapping once when the examiner taps twice and tapping twice when the examiner taps once. Ten trials are completed (five with a single tapping and five with a double tapping); single and double tapings are intermixed in a fixed order. *Subtest 5, Go/No-Go Task*: the same alternating signals used in the previous

‘Conflicting Instructions’ subtest, but the participant is required to refrain from tapping when the examiner taps twice and to tap once when the examiner taps once. Scores were calculated based on number of mistakes within both subtests, with a total possible score of three (0 mistakes = 3, 1 mistake = 2, ≤ 2 mistakes = 0).

Alcohol Intoxication Measures

Biphasic Alcohol Effects Scale (Martin, Earleywine, Musty, Perrine, & Swift, 1993). The BAES is a self-report scale used to assess subjective feelings of *sedation* and *stimulation* effects of alcohol consumption. Participants rate the extent to which they are currently experiencing feelings described by seven sedation adjectives (e.g., ‘heavy head’, ‘inactive’) and seven stimulation adjectives (e.g., ‘energised’, ‘excited’) on an 11-point Likert type scale, with response options ranging from 0 = ‘not at all’ to 10 = ‘extremely’. Responses are summed to form total Sedation and Stimulation subscales scores, with higher scores indicative of greater sedation and stimulation, respectively. The BAES has been shown to have high internal consistency for both subscales (Cronbach’s alpha = .85 to .94, respectively) and the validity of the subscales has been supported by factor analysis (Earleywine & Erblich, 1996). The BAES is typically administered prior to beverage administration and repeated 3-4 more times after consumption to assess effects of alcohol on both the ascending and descending blood alcohol limbs (Rueger & King, 2013).

Beverage Rating Scale (BRS); (Fillmore & Vogel-Sprott, 2000). The BRS was administered at the end of the study to establish whether participants could distinguish if they had received placebo or alcoholic beverages. The BRS is regularly used as a manipulation check in alcohol experimental research (Marczinski & Fillmore, 2003; Peacock, Bruno, Martin, & Carr, 2013). Participants report the perceived alcoholic content of the beverage, on a scale of 0 to 10 bottles of beer

(containing 4.8% alcohol) they think they would have consumed to reach the peak level of intoxication perceived during the session.

Breath Alcohol Concentration (BrAC): BrACs were determined from breath samples measured by a breathalyser provided and calibrated by Tasmania Police, model Lion SD-400 Alcometer®.

Experimental Measures

Flanker Task (Eriksen & Eriksen, 1974). The Flanker task is a computer based categorisation task of four target letters (H, K, S, C) that appear above a fixation cross at the centre of the screen. Letters appear either alone or flanked by other noise letters (flankers). Participants are required to respond to the central target by pressing a spatially compatible button on the keyboard while ignoring the flanker 'noise' letters. Target letters H or K require the participant to press Q at the left side of the keyboard. Target letters S or C require a press of the P key at the right of the keyboard. On congruent trials, flankers are compatible (e.g. SSSSS or SSCSS); on incongruent trials, flankers are incompatible (e.g. SSKSS or SSHSS). There are 200 congruent trials and 200 incongruent trials. Participants press the spacebar to start each new trial. Stimuli are presented for 1000ms. Approximate total task length is 12 minutes. Participants are encouraged to be as fast and as accurate as possible. The flanker task provides a measure of the ability to quickly respond to task-relevant attributes of stimuli (target letter) and suppress responses to task-irrelevant stimuli (flankers). This task was conducted using the computer program Inquisit 5 by Millisecond Software.

Cued Go/No-Go Task (as described in Fillmore, Marczinski, & Bowman, 2005; Marczinski & Fillmore, 2003; Fillmore, Rush, & Hays, 2006). This Go/No-Go task provides a measure of response inhibition by rapid establishment of cue-

dependence via presentation of cues highly likely to be followed by the expected target, the probability of which is then reduced, requiring alteration of the activated response. Presentation of a green rectangle requires a motor response ('go' response; press of space bar on computer keyboard) whereas a blue rectangle requires the participant to withhold a response ('no-go'). Preliminary cues of vertical or horizontal blank rectangle cues signal which colour will be presented next, developing cue-dependence for activational or inhibitory mechanisms. Preliminary cues are displayed for 800ms, inter-stimulus interval is 500ms, target cue displayed for 1000ms and the inter-trial interval is 700ms. There are 250 trials consisting of 100 vertical cue 'Go' targets and 25 vertical cue 'No-Go' targets, and 100 horizontal cue 'No-Go' targets and 25 horizontal cue 'Go' targets. Failure to inhibit responses to no-go targets is more frequent following go cues compared with no-go cues (Fillmore, Rush, & Hays, 2006). The no-go condition is also highly sensitive to the effects of alcohol. This task was conducted using the computer program Inquisit 5 by Millisecond Software and was approximately 10 minutes in length.

Social Disinhibition Task (SDT; Honan, Allen, Fisher, Osborne-Crowley, & McDonald, in press). The SDT is a recently developed measure of social disinhibition that has recently been validated for use in individuals with traumatic brain injury and will be used for the first time in this study in an alcohol intoxicated sample. Participants are asked to view scenes of complex social situations portraying either salient (non-faux pas) or subtle (faux pas) negativity while being told a brief description about the scene. Participants must say the first word that comes to mind to describe a particular character in the scene.

The task consists of three parts, each with 10 items (five faux-pas, five non-faux pas). *Part A* (control task) requires participants to say the first word that comes

to mind following each item presentation. In *Part B* (inhibition task), the participant is required to describe a particular character after being directed not to say anything negative or anything that is likely to offend the person, or comment on the person's age, size, race, ethnicity or religion. This task requires participants to inhibit automatic negative responses. In *Part C* (guided response task), participants are directed to say the first word that comes to mind that is positive, and not negative, to describe a particular character. Part C therefore requires both inhibition of negative responses and the generation of more socially acceptable, positive utterances in response to a specific request to do so.

In the non-faux pas items, the intentions of the characters are explicit (e.g., a young woman is visibly upset with her boyfriend for forgetting her birthday). Faux pas items contain more subtle negativity that require higher level ToM ability to process, due to the absence of explicit negativity in both the body language in the pictorial scene and in the experimenter's verbal description of the scene (e.g. a scene of a boss who stands by happily while his female assistant struggles to carry a stack of files). Items are scored as: 0 = negative response, 1 = neutral response, 2 = positive response. Each part is scored out of 10, calculated as the average of the total faux pas and total non-faux pas scores. Where positive utterances by the participant were accompanied by contradictory body language or tone of voice (e.g. eye-roll and contradictory tone) the item was scored as negative rather than as positive. Inter-rater reliability was good ($\kappa = .82$). Sample items for this task are displayed in Appendix F.

Procedure

All participants were required to fast from food for four hours prior to participation, but consume two pieces of toast one hour prior to attending a 150-

minute experimental session at the University of Tasmania. They were also required to abstain from caffeine for eight hours prior to the session. Remuneration for participation was offered in the form of one Village Cinema movie ticket, or first year psychology students could opt for three hours of research participation credit. Following provision of informed consent, the TLFB was completed as a final eligibility check to ensure participants had consumed at least two standard drinks in one session within the previous fortnight. All participants were breathalysed to ensure a baseline BrAC of .000%. A 150ml placebo beverage consisting of 10ml Schweppes® lime syrup, three drops Angostura® aromatic bitters, and 137ml of soda water, with 3ml vodka (Smirnoff Red Label®, No. 21) floated on top and a light mist of vodka sprayed into the cup to create a strong odour of alcohol (Peacock et al., 2013) was administered prior to baseline assessments to maintain consistent alcohol expectancies across all tasks. Baseline assessments (BAES and the FAB) were completed.

Participants were then administered a treatment beverage according to the allocated condition. The alcohol condition consumed a 750ml beverage that comprised 90ml Schweppes® lime syrup, 5ml Angostura® aromatic bitters, 300ml soda water and a dose of vodka (Smirnoff Red Label®, No. 21), mixed with still water to make up 750ml. The vodka dose was calculated using the Widmark formula (Dry et al., 2012) (see Appendix E) for each participant to reach a target BrAC of .080% at 60 minutes post consumption. The placebo treatment beverage comprised 90ml Schweppes® lime syrup, 5ml Angostura® aromatic bitters, 300ml soda water and 305ml still water. The lime syrup and bitters were used to make the beverage more palatable to aid in quick consumption, and to mask the taste and smell of alcohol (and lack thereof in the placebo condition). A stopwatch was provided to

enable participants to pace their drinking and to consume the beverage it at a steady rate over a 10 minute period. A 50 minute absorption period followed during which time participants watched a neutral video (David Attenborough's Great Barrier Reef, Australian Broadcasting Company, 2016).

BrACs were recorded following the absorption period. The BAES was administered a second time and a 15 minute emotion recognition task was completed as part of a separate study before commencing the test protocol of the present study. Participants were therefore breathalysed approximately 65 minutes after consumption of the beverage, at which point they were expected to have reached the target BrAC of .080%. Participants immediately commenced the SDT, followed by the flanker task, then the GNG task. BrAC readings were taken prior to commencement of each task and at completion of the test protocol. The BAES was completed a third time at the conclusion of testing, followed by the Beverage Rating Scale. Participants were debriefed, and those in the alcohol condition were provided with food and entertainment in a comfortable environment during the detoxification period. Participants were released from the care of the researchers once they recorded two consecutive BrACs of .030 or less, 15 minutes apart (or .000% if they held a provisional drivers' licence if they intended to drive).

Design

This study was a mixed factorial, single-blind, placebo-controlled, quasi-randomised sex-block design.

Statistical Analyses

Data was analysed using IBM SPSS Statistics Version 22.0. Data was screened for violations of assumptions of normality and homogeneity of variance. Independent samples *t*-tests were conducted to examine differences between groups

in demographic, eligibility and baseline characteristics. Data for the BAES had non-normal positive skew. A square root transformation normalised the data however this did not impact on the results. Therefore results reported here are from the raw data.

For the SDT, a 2 (condition) \times 3 (part) \times 2 (item type) mixed ANOVA identified a violation of Levene's test of homogeneity of variance on a single component of the SDT (Part A, faux pas items; $p = .038$). To address this problem, a mixed model full information maximum likelihood (FIML) approach was taken to examine group differences in performance and response latency. The mixed model FIML approach is suitable in the presence of violations to data assumptions, and therefore enables a more robust analysis than conventional methods (Enders, 2011). As this study was specifically examining and comparing group performance within each part and within each item type in each part, only results relating to these aims are reported. The main effect for group, and group by item type interaction were not pertinent to the current study and are not reported.

There was severe positive skew for all SDT response latency variables. Inverse transformations were performed as recommended by Tabachnick and Fidell (2007) on all response latency variables and analyses re-conducted. The transformation reduced the skew, however, there was no statistical improvement to results. Therefore, for simplicity and ease of interpretation, the results of the analysis using the raw data only are reported. When interpreting SDT results, higher scores indicate more positive responses, and lower scores indicates more negative responses.

Due to technical malfunction during electronic administration of the Flanker task and Go/No-Go Tasks, three participants (two placebo, one alcohol) had missing

data (i.e., $n = 61$ for these tasks). The error rate statistics for the Flanker task were severely positive skewed. A logarithmic transformation was performed with a constant (+2) added to ensure the transformation was performed correctly (i.e. values less than 1 are problematic when performing a logarithmic transformation) as recommended by Tabachnick and Fidell (2007). The transformation reduced the skew but did not impact on the results, therefore analyses were conducted using raw data. For the Go/No-Go task, only overall reaction time data (congruent and incongruent combined) was recorded by Inquisit. The error rate data for this task was moderately skewed, however, logarithmic transformation did not substantially alter results, so independent t -tests were conducted on the raw data.

Assumptions for all other analyses were met. Spearman's non-parametric correlations were conducted to examine the relationship between Part A (control task) and Part B (inhibition task) scores on the SDT, and flanker and GNG tasks. Correlations were interpreted in terms of the size of the correlation according to the recommendations of Cohen (1988), where .10 is indicative of a small effect, .25 is indicative of a moderate effect, and .50 is indicative of a large effect. Cohen's d or partial eta-squared (η^2) effect sizes are reported and interpreted in accordance with Cohen's (1988) recommendations. For Cohen's d , .20 indicates a small effect, .50 a moderate effect, and .80 indicates a large effect. Partial eta-squared is interpreted as .01 as a small effect, .09 as a medium effect, and .25 as a large effect. Reaction Time data is reported in seconds.

Results

Eligibility and Baseline Measures

No significant differences were detected between groups for eligibility measures including the AUDIT, K10 and TLFB. Groups did not differ in baseline

inhibitory control ability, indicated by performance on the FAB. See Table 2 for descriptive data and comparison statistics for these analyses.

Table 2.

Comparison of Group Characteristics on Eligibility Measures

	Alcohol		Placebo		<i>t</i> (62)	Sig	95% CI's		<i>d</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>			Lower	Upper	
AUDIT	6.65	3.38	5.70	4.80	1.11	.270	-.76	2.65	.28
K10	13.94	3.47	14.21	3.09	-.34	.737	-1.92	1.36	.08
TLFB	19.04	17.0	16.79	15.15	.56	.577	-5.78	10.29	.14
FAB	2.94	.25	2.85	.51	.86	.392	-.12	.30	.39

Note: AUDIT = Alcohol Use Disorders Identification Test; FAB = Frontal Assessment Battery Conflicting Instructions and Go/No-Go combined scores; K10= Kessler Psychological Distress Scale; TLFB = Timeline Followback.

Alcohol Intoxication Measures

Breath Alcohol Concentrations and Subjective Intoxication Measures

No detectable BrAC's were observed in the placebo condition. At 65 minutes post-consumption, immediately prior to the administration of the SDT, participants in the alcohol condition recorded a mean BrAC of .077% (*SD* = .020). Prior to the flanker task, approximately 75 minutes post-consumption, mean BrAC was .074% (*SD* = .019). Prior to the GNG, approximately 90 minutes post-consumption, mean BrAC was .070% (*SD* = .016). The mean BrAC was .066% (*SD* = .016) at conclusion of the test battery, approximately 100 minutes post consumption.

Biphasic Alcohol Effects Scale

Figure 1 displays the mean BAES stimulation and sedation subscale change scores according to treatment condition. There was a 2 Condition \times 2 Subscale

(Sedation, Stimulation) \times 3 Time (baseline, following absorption, after GNG) interaction, $F(4, 320) = 8.42, p < .001, r = .16$. Post-hoc comparisons indicated that at baseline, subjective ratings of both subscales of sedation and stimulation were similar between groups, $M_{\text{Diff}} = .25, p = .558, CI_{\text{Diff}} [-.58, 1.07], d = .15$. At time two, the alcohol group reported higher levels of sedation, $M_{\text{Diff}} = .96, p = .022, CI_{\text{Diff}} [.14, 1.79], d = .59$, and stimulation, $M_{\text{Diff}} = .85, p = .043, CI_{\text{Diff}} [.03, 1.67], d = .50$ than the placebo group. At time three, alcohol reported significantly higher levels of sedation, $M_{\text{Diff}} = 1.05, p = .013, CI_{\text{Diff}} [.22, 1.67], d = .63$, but stimulation scores were not different between groups $M_{\text{Diff}} = .41, p = .325, CI_{\text{Diff}} [-.41, 1.24], d = .24$.

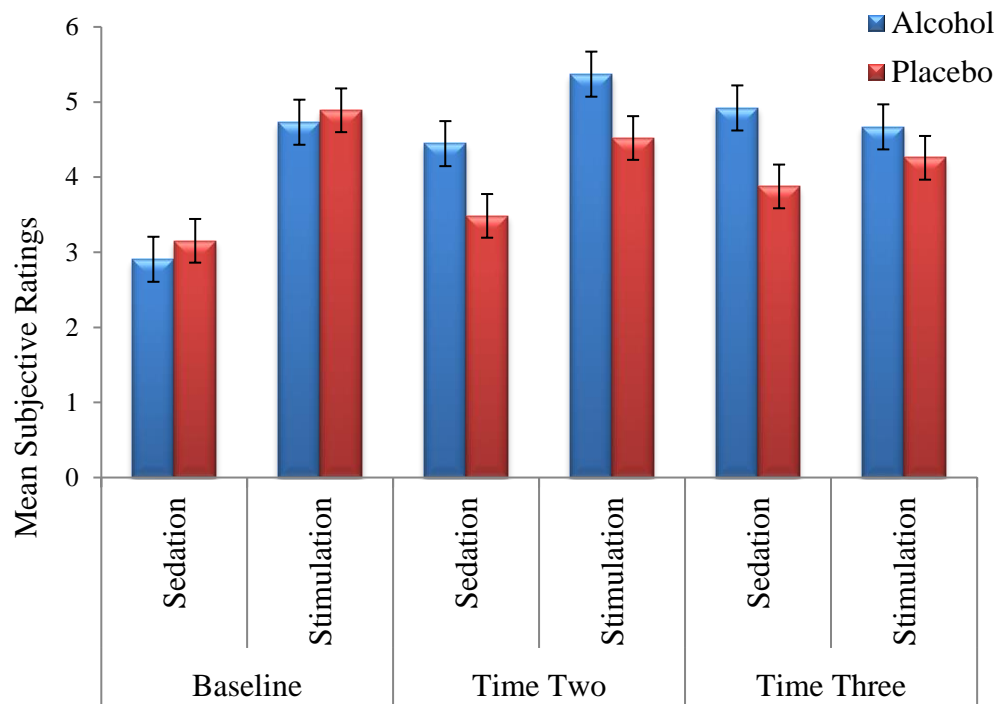


Figure 1. Subjective ratings of effects of alcohol on sedation and stimulation for each group at each time point. Error bars represent standard error values.

Beverage Rating Scale

An independent samples *t*-test indicated there was a significant difference between groups in perceived alcohol content of the beverages administered, $t(62) = 9.61, p < .001, d = 1.84$, with greater alcohol intake reported by participants in alcohol ($M = 4.40, SD = 1.11$) relative to placebo ($M = 1.48, SD = 1.31$) conditions. All participants in the alcohol condition and 25 of the 33 (approximately 76%) participants in the placebo condition reported that the treatment beverage contained alcohol.

Experimental Measures

Measure of Attentional Inhibition: Flanker Task

A $2 \text{ (Group)} \times 2 \text{ (Compatibility)}$ mixed design ANOVA was performed to examine differences in error rates for compatibility of target types and between conditions. The results of this analysis is depicted in Figure 2. There was a main effect of group [$F(1, 59) = 4.11, p = .047, \eta^2 = .07$], with alcohol intoxicated participants ($M = .15, SD = .08$) recording significantly higher error rates overall than those administered a placebo ($M = .10, SD = .08$). The overall rate of error was higher following incompatible targets ($M = .13, SD = .09$) than compatible targets ($M = .11, SD = .08$), indicated by a significant main effect of compatibility [$F(1, 59) = 16.73, p < .001, \eta^2 = .22$]. No group by compatibility interaction was found [$F(1, 59) = .01, p = .905, \eta^2 = .00$].

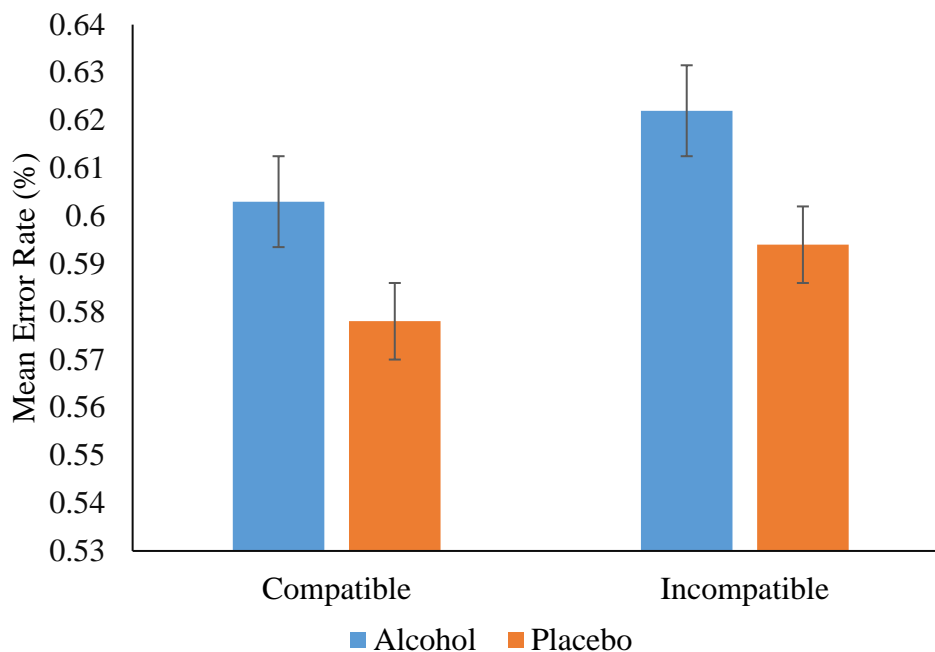


Figure 2. Rate of error on the Flanker task by compatibility. Error bars represent standard error values.

A second 2 Group \times 2 Compatibility mixed ANOVA was conducted to determine if reaction time in the flanker task varied according to compatibility of type across groups. The results of this analysis is depicted in Figure 3. There was a trending main effect of group on reaction time [$F(1, 59) = 3.93, p = .052, \eta^2 = .06$] toward the alcohol group ($M = .62$ seconds, $SD = .05$) being slower than the placebo group ($M = .59$ seconds, $SD = .05$), although this was a small to moderate effect ($\eta^2 = .06$). A significant main effect of compatibility [$F(1, 59) = 88.66, p < .001, \eta^2 = .60$], demonstrated that overall reaction time was significantly slower following incompatible targets ($M = .59, SD = .05$) than compatible targets ($M = .61, SD = .05$). There was no interaction effect of group and target type, $F(1, 59) = .66, p = .419, \eta^2 = .011$.

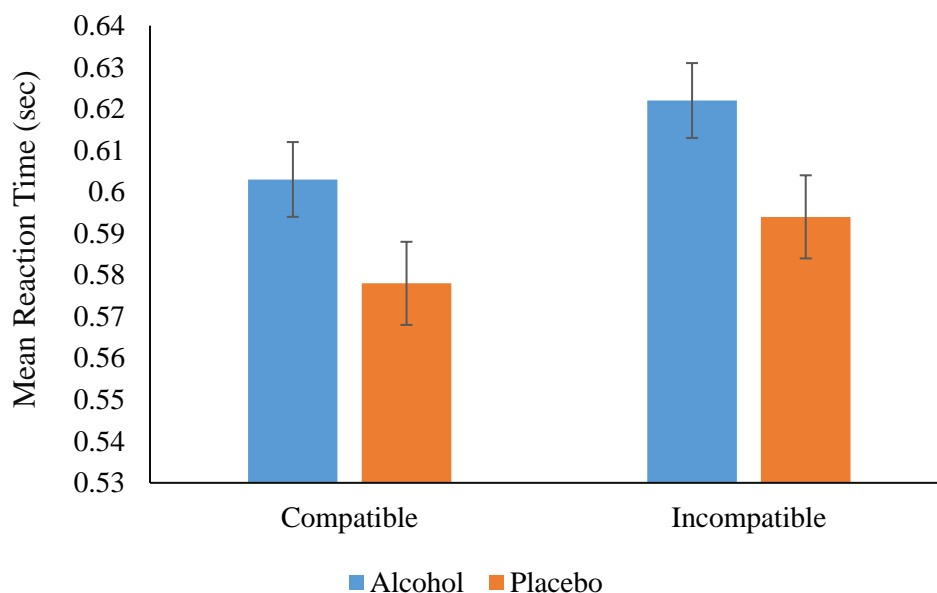


Figure 3. Mean reaction time as a function of compatibility and treatment group. Error bars represent standard errors.

Measure of Response Inhibition: Go/No-Go Task

T-test analyses of the cued Go/No-Go task did not identify any group differences in errors of omission (neglecting to press the spacebar at presentation of a ‘Go’ target), $t(59) = .42$, $M_{\text{Diff}} = .02$, $p = .675$, $CI_{\text{diff}} [-.13, .08]$, $d = .11$. There were also no group differences in inhibitory failures (quantified by presses of the spacebar following a ‘No-Go’ target), $t(59) = .46$, $M_{\text{Diff}} = .02$, $p = .646$, $CI_{\text{diff}} [-.13, .08]$, $d = .12$. Additionally, no group differences were detected for overall reaction time to targets, $t(59) = .451$, $M_{\text{Diff}} = 5.33$, $p = .654$, $CI_{\text{diff}} [-18.34, 29.01]$, $d = .12$.

Performance on the SDT

The FIML mixed models analysis of the SDT data revealed a significant main effect of Part, $F(2, 320) = 214.79$, $p < .001$, $r = .63$. Post-hoc comparisons indicated that, overall, participants responded more negatively (i.e. produced more negative than positive utterances) in Part A (control trial; $M = 1.96$, $SD = 1.56$) than both Part

B (inhibition trial; $M = 5.14$, $SD = 1.56$), $M_{\text{Diff}} = 3.18$, $p < .001$, $CI_{\text{Diff}} [-3.65, -2.71]$, $d = 1.82$, and Part C (guided response trial; $M = 6.84$, $SD = 1.56$), $M_{\text{Diff}} = 4.88$, $p < .001$, $CI_{\text{Diff}} [-5.35, -4.41]$. Responses in Part C were significantly more positive than Part B, $M_{\text{Diff}} = 1.70$, $p < .001$, $CI_{\text{Diff}} [1.23, 2.18]$, $d = .98$. No 2 Group \times 3 Part interaction [$F(2, 320) = .04$, $p = .964$, $r = .10$] was detected.

The analysis also revealed a significant 2 Group \times 2 Item Type \times 3 Part interaction, $F(4, 320) = 3.11$, $p = .016$, $r = .01$. The results of this analysis is depicted in Figure 4. Post-hoc group comparison analyses indicated that those who consumed alcohol gave significantly more negative responses to faux pas items than the placebo group, on both Part A, $M_{\text{Diff}} = 1.06$, $p = .041$, $CI_{\text{diff}} [.04, 2.07]$, $d = .54$, and Part B, $M_{\text{Diff}} = 1.11$, $p = .032$, $CI_{\text{diff}} [.09, 2.12]$, $d = .56$. However, no group differences were detected for faux pas items in Part C, $M_{\text{Diff}} = .52$, $p = .312$, $CI_{\text{diff}} [-.49, 1.54]$, $d = .26$. No significant differences were detected between groups for non-faux pas items on Part A ($M_{\text{Diff}} = .12$, $p = .823$, $CI_{\text{diff}} [-.90, 1.13]$, $d = .05$), Part B ($M_{\text{Diff}} = .09$, $p = .857$, $CI_{\text{diff}} [-.92, 1.11]$, $d = .05$), or Part C ($M_{\text{Diff}} = .53$, $p = .306$, $CI_{\text{diff}} [-.49, 1.54]$, $d = .25$).

Additional post-hoc analyses for the 3-way interaction indicated that in Part A, there were no significant differences in scores between faux pas and non-faux pas items in the alcohol group ($M_{\text{Diff}} = .36$, $p = .465$, $CI_{\text{diff}} [-.60, 1.31]$, $d = .17$) or the placebo group ($M_{\text{Diff}} = .82$, $p = .083$, $CI_{\text{diff}} [-.11, 1.74]$, $d = .40$), although the small to moderate effect size suggests there was a possible trend toward placebo participants providing more positive responses to faux pas items than non-faux pas items.

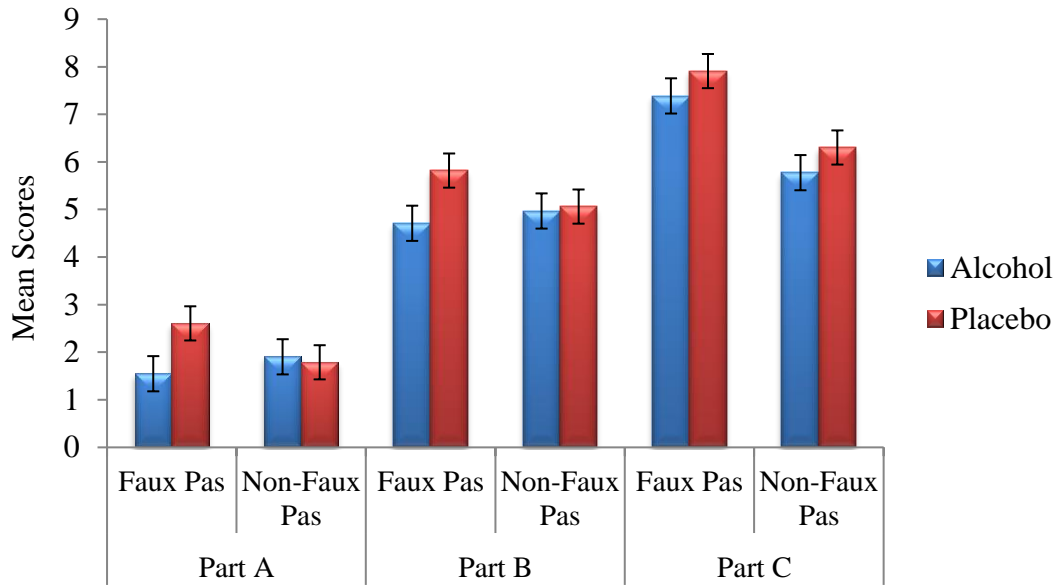


Figure 4. Responses to Item Type and each Part stratified by condition. Higher scores are indicative of more positive utterances. Error bars represent standard errors.

In Part B there were no significant differences in scores between item types within the alcohol group ($M_{\text{Diff}} = .26, p = .595, CI_{\text{diff}} [-.70, 1.21], d = .13$) or within the placebo group ($M_{\text{Diff}} = .76, p = .108, CI_{\text{diff}} [-.17, 1.68], d = .37$). However, in Part C, participants provided significantly more positive responses to faux pas items than non-faux pas items in both the alcohol group ($M_{\text{Diff}} = 1.61, p = .001, CI_{\text{diff}} [.66, 2.57], d = .78$) and the placebo group ($M_{\text{Diff}} = 1.61, p = .001, CI_{\text{diff}} [.68, 2.53], d = .78$).

Paired samples *t*-tests were also conducted to compare change between SDT parts within each item type in each group. Significantly more positive responses were provided to faux pas items in Part B than Part A in both alcohol [$t(30) = 7.43, p < .001, d = 1.68$] and placebo [$t(32) = 5.77, p < .001, d = 1.37$], and participants were more positive to faux pas items in Part C than Part B in both the alcohol [$t(30)$

= 5.02, $p < .001$, $d = 1.25$] and the placebo [$t(32) = 5.03$, $p < .001$, $d = .97$] groups.

For non-faux pas items, participants were more positive in Part B than Part A in the alcohol group [$t(30) = 5.65$, $p < .001$, $d = 1.50$] and in the placebo group [$t(32) = 5.65$, $p < .001$, $d = 1.49$]. The placebo condition also produced significantly more positive responses to non-faux pas items in Part C than in Part B [$t(32) = 2.54$, $p = .016$, $d = .49$], however, participants who had consumed alcohol did not change the positivity of responses to non-faux pas items from Part B to Part C [$t(30) = 1.4$, $p = .168$, $d = .34$].

Correlations Between SDT Inhibition Scores, the Flanker and GNG

Correlations between Parts A (control task) and B (inhibition task) of the SDT and the Flanker and GNG tasks are presented in Table 3. On Part A, small-moderate and moderate negative correlations were found between scores on faux pas items and error rates on both the flanker and GNG task for the alcohol group. For the placebo group, there were small to negligible correlations between Part A faux pas scores and the GNG task, however, moderate positive correlations were present between error rates related to both compatible and incompatible target types on the Flanker task. A small-moderate correlation was found between non-faux pas scores and error rate following incompatible flanker targets, and a moderate correlation was found for non-faux pas items and error rate to incompatible flanker targets. Only negligible or small correlations were present between all Part B scores and GNG and Flanker error rates for both conditions.

Table 3.

Correlations Between SDT Scores For Item Type (Parts A & B) and the Go/No-Go and Flanker Tasks

	Go/No-Go		Flanker	
	Overall Error Rate	Commission Error Rate	Error Rate Compatible	Error Rate Incompatible
Part A				
Faux Pas				
Alcohol	-.294	-.384	-.200	-.229
Placebo	.055	.042	.398	.314
Non Faux Pas				
Alcohol	-.123	-.199	.071	.057
Placebo	.159	.090	.316	.238
Part B				
Faux Pas				
Alcohol	.078	.024	.051	.055
Placebo	-.088	-.091	-.101	-.166
Non Faux Pas				
Alcohol	.109	.017	-.111	.020
Placebo	-.125	-.128	-.001	-.150

Note: Correlations greater than $r = .20$, indicating at least a small to moderate effect size, are highlighted in bold text (Cohen, 1988).

Response Latency on the SDT

The FIML mixed models analysis did not reveal any differences between conditions in response latency on the SDT, with no 2 Group \times 3 Part interaction [$F(4, 320) = 1.08, p = .367, r = .06$], no 2 Group \times 2 Item Type interaction [$F(2, 320) = 1.93, p = .147, r = .08$], and no 2 Group \times 2 Item Type \times 3 Part interaction [$F(4, 320) = .55, p = .700, r = .04$].

Discussion

The primary aim of this study was to examine the effects of alcohol on the ability to inhibit automatic negative verbal responses to negative social information, and whether the ability to inhibit was dependent on ToM ability. A secondary aim was to examine whether intoxicated individuals could adjust their responses to provide more positive utterances when specifically requested to do so. The results of baseline assessments indicated that group characteristics were homogeneous, and the subjective and objective measures of intoxication demonstrated that the treatment manipulation was effective, ascertaining that differences between groups on experimental tasks can be attributed to treatment manipulation.

In replication of the findings of the prior literature, it was hypothesised that alcohol intoxicated individuals perform more poorly on laboratory-based inhibition tasks. In support of this hypothesis, intoxicated individuals did record significantly higher error rates overall than the placebo group on the Flanker task. This suggests that alcohol intoxication did impair *inhibitory control* on this task. However, there was a lack of group difference found on the GNG task indicates that *response inhibition* was not impaired. This finding is inconsistent with prior literature where reduced performance in alcohol intoxicated individuals (relative to a placebo) has been found on both the ascending and descending limbs of the blood alcohol curve, at an approximate BrAC of .060% (e.g., Fillmore & Vogel-Sprott, 2000; Ostling & Fillmore, 2010; Fillmore & Weafer, 2012). There was also some indication of increased reaction time in alcohol-intoxicated individuals compared to placebo individuals. Specifically, while there was a trend toward slower reaction times in the flanker task ($p = .052$) for the alcohol-intoxicated individuals, similar reaction times were found between groups on the GNG task. Given the GNG task was administered

last in the current test battery, it is possible that this lack of between-group difference is attributable to recovery from intoxication. Indeed, prior findings in the literature do indicate that reaction time usually improves during alcohol descent (Weafer & Fillmore, 2012).

For the SDT, evidence of task efficacy was demonstrated by the tendency of all participants to provide negative responses in Part A (the control task), more positive responses in Part B (the inhibition task), and a higher rate of positive responses in Part C (the guided response task). More specifically, the results demonstrate that both groups were able to follow task instructions to inhibit the production of negative responses in Part B, and to adjust responses as directed (i.e., to produce a positive response) in Part C. Intoxicated individuals' ability to provide more positive responses in Part B in comparison to Part A demonstrates an ability to inhibit negative utterances to negative social information. However, upon further examination of this effect, it appeared that specific differences were apparent if item-type was considered. Specifically, while there were no between group differences in response to non-faux pas items in both Parts A and B, intoxicated individuals were significantly more negative to the faux pas items in both Parts A and B, than the placebo group.

Based on the AMM paradigm (Steele & Josephs, 1990), it was hypothesised that the alcohol group would have difficulty detecting negativity in faux pas items and would thus provide more positive responses to items requiring ToM. This hypothesis was not supported. As previously indicated, both groups responded similarly to non-faux pas items in each part, but the alcohol group responded more negatively to faux pas items in Parts A and B than the placebo group. This is of particular interest, as according to AMM, alcohol narrows attentional capacity which

results in only salient information being processed (Steele & Josephs, 1990). As negativity in the faux pas items is not considered salient and requires the implementation of higher order social cognitive ToM ability to process, it was expected that intoxicated individuals would be less able to identify the negativity in the scene and thus respond more positively to faux pas items.

A possible explanation for this converse finding is that alcohol may actually enhance ToM ability at moderate doses of alcohol. Important findings by McDonald et al. (2014) describe difficulties for people with traumatic brain injury (TBI) to inhibit self-referential thoughts in order to consider another person's point of view. McDonald et al. suggest this difficulty is due to the high cognitive load involved in simultaneously inhibiting and processing information. In line with this, it is possible that alcohol facilitates ToM by reducing cognitive resources needed to inhibit self-referential thoughts, enabling intoxicated individuals to attend to external information, particularly when that information is salient. The SDT directs participants to consider another person's viewpoint, rather than activating self-referential cognitions. Therefore, the instruction to consider another person's viewpoint is the salient cue in this instance so intoxicated individuals are better able to attend to that information. A recent study by Dolder et al. (2016) found that alcohol enhanced affective ToM for positive emotional stimuli. This suggests that, contrary to Mitchell et al.'s (2011) findings that alcohol impaired ToM performance in intoxicated individuals, ToM ability may actually be enhanced. Additionally, the measures used by Mitchell et al. to assess ToM have questionable construct validity (Johnston, Miles, & McKinlay, 2008), or are influenced by high verbal loading.

The performance of alcohol intoxicated individuals on the SDT task also demonstrated that when provided with more specific guidance about how to respond,

intoxicated individuals were able to adjust their responses and provide more socially acceptable, positive responses to the negative social information. In Part C of the SDT, participants in both groups were more positive to faux pas items than to non-faux pas items. This demonstrates that both groups found it easier to provide positive responses on the guided response trial. This effect is especially pertinent for the alcohol group, as they were significantly more negative to faux pas items on Parts A and B in comparison to the placebo group, yet in the guided response trial, intoxicated individuals were able to match the placebo group in terms of positivity of responses. This finding is consistent with prior literature that suggests that although alcohol may impair the ability to inhibit a response, behaviour can still be guided by the consequences of that behaviour (Fillmore & Vogel-Sprott, 2000).

In terms of AMM, these results may suggest that in contexts where social expectations are meaningful enough to an intoxicated individual for them to be concerned about their performance/behavioural consequences, the provision of a set of response guidelines may successfully guide behaviour. Intoxicated individuals appear to be able to override the effects of alcohol to inhibit an inappropriate social behaviour. For example, in the context of this experiment, it may be that the participant was concerned that they would not perform as well as other participants on the tasks. Therefore, the pressure to perform represents a salient internal cue, which, when provided with clear response guidelines and social expectations, assists the participant to more easily inhibit and adjust their response. The finding that intoxicated individuals are able to adjust their social behaviour when provided clear guidelines, even under the influence of a moderate to high dose of alcohol, may be useful information for development of social policy or when considering criminal cases where alcohol intoxication is involved.

Small to moderate negative correlations between Part A of the SDT and the Flanker task, and between the SDT and the GNG task suggest that a higher rate of negative responses were associated with greater error rates. As negative responses on the SDT represent reduced inhibition, and Part A does not require implementation of inhibitory control, this negative correlation with inhibitory errors on laboratory tasks is expected. For the placebo group, there was a moderate positive correlation to both incompatible and compatible targets on the Flanker task. This suggests that positive responses on Part A of the SDT is associated with a higher error rate, indicating that participants who provide more negative responses make less errors, because they are not inhibiting their automatic negative response.

The negligible correlations for both Part B of the SDT and the Flanker, and Part B and the GNG may suggest the two laboratory measures are unable to predict the ability to inhibit automatic social responses in a context where social restrictions are in place, for either the alcohol group or the placebo group. This further supports the findings that intoxicated individuals can override alcohol related impaired inhibitory control if required to. It also remains possible that the SDT task may lack the sensitivity to detect inhibition ability in intoxicated individuals. However, this seems unlikely as intoxicated participants were able to change their responses from Part A to Part B, providing a clear indication of an inhibitory effect.

A limitation of this study may be that alcohol expectancies influenced behaviour (Hull & Slone, 2004; Steele & Josephs, 1990). However, it appeared that the level of doubt in both groups as to whether they were administered alcohol or not was consistent across groups, with placebo participants tending to estimate they had consumed alcohol, regardless of estimations of a smaller dose. Additionally, frequent comments were made by participants in the alcohol group regarding concern

over potential embarrassment if they were to report having consumed alcohol but had actually been administered placebo. This could be considered additional evidence of restraint on behaviour stemming from alcohol expectancies.

Second, scoring for the SDT task assumes that negative responses are socially inappropriate, with lower scores indicating more negativity. The SDT is designed to assess the ability to inhibit negative responses to negative social information.

However, in real life, provision of a positive response to some of the situations may not be considered an appropriate response. For example, displaying disapproval to deter inappropriate behaviour exhibited by someone else may actually be an effective and adaptive social behaviour. Nevertheless, the SDT provides an indication of the ability to inhibit responses to social information, and overall participants were able to change their responses in each part.

Additionally, results on the GNG task may have been different if the experimental battery was counterbalanced. However, counterbalancing was not implemented for two reasons. The first reason was to limit the amount of noise in the data to aid in simple interpretation. Second, it was important to assess the SDT at peak BrAC as higher alcohol concentrations are associated with negative social outcomes (Morgan & McAtamney, 2009), which may be related to social disinhibition.

The results of this study demonstrate that alcohol intoxicated individuals are able to inhibit negative responses to negative social information, but are less able to inhibit negative responses to social information requiring ToM ability. It also shows that people under the influence of a moderate to high dose of alcohol can adjust their responses when provided with specific guidelines on how to respond. Further research could investigate the effect of a higher dose of alcohol on performance on

SDT, as it is possible that as alcohol concentration increases, cognitive capacity will also decrease so that subtle information is not processed. Research should also aim to extend the content of the SDT, particularly integrating items into the task that portray different cultural and social contexts (e.g., those with higher social constraints compared to those with few constraints). This would enable examination of the ability to identify subtle negative information in a range of contexts, and the ability to provide socially acceptable responses according to the context. The findings of this study extend current understandings of the mechanisms involved in negative social behaviours following acute alcohol administration. Understanding why intoxicated people respond negatively in social contexts may have implications for the development of public policy and understanding alcohol related crimes.

References

- Abroms, B. D., Fillmore, M. T., & Marczynski, C. A. (2003). Alcohol-induced impairment of behavioral control: effects on the alteration and suppression of prepotent responses. *Journal of Studies on Alcohol*, 64, 687-695.
doi:10.15288/jsa.2003.64.687
- Arciniegas, D. B., & Wortzel, H. S. (2014). Emotional and behavioral dyscontrol after traumatic brain injury. *Psychiatric Clinics of North America*, 37, 31-53.
doi:10.1016/j.psc.2013.12.001
- Arnaud, B., Malet, L., Teissedre, F., Izaute, M., Moustafa, F., Geneste, J., ... & Brousse, G. (2010). Validity Study of Kessler's Psychological Distress Scales Conducted Among Patients Admitted to French Emergency Department for Alcohol Consumption-Related Disorders. *Alcoholism: Clinical and Experimental Research*, 34, 1235-1245. doi:10.1111/j.1530-0277.2010.01201.x
- Assaad, J. M., Pihl, R. O., Seguin, J. R., Nagin, D. S., Vitaro, F., & Tremblay, R. E. (2006). Intoxicated behavioral disinhibition and the heart rate response to alcohol. *Experimental and Clinical Psychopharmacology*, 14, 377-388.
doi:10.1037/1064-1297.14.3.377
- Attwood, A. S., & Munafò, M. R. (2014). Effects of acute alcohol consumption and processing of emotion in faces: Implications for understanding alcohol-related aggression. *Journal of Psychopharmacology*, 28, 719-732. doi: 10.1177/0269881114536476
- Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., & Monteiro, M. G. (2001). *Audit. The Alcohol Use Disorders Identification Test (AUDIT): Guidelines for use in primary care*. World Health Organisation. Geneva: Switzerland.

- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The “Reading the Mind in the Eyes” test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry*, 42, 241-251. doi: 10.1111/1469-7610.00715
- Bibby, H., & McDonald, S. (2005). Theory of mind after traumatic brain injury. *Neuropsychologia*, 43, 99–114. doi:10.1016/j.neuropsychologia.2004.04.027
- Bramham, J., Morris, R., Hornak, J., Bullock, P., & Polkey, C. (2009). Social and emotional functioning following bilateral and unilateral neurosurgical prefrontal cortex lesions. *Journal of Neuropsychology*, 3, 125-143. doi: 10.1348/174866408X293994
- Burden, M. J., Jacobson, J. L., Westerlund, A., Lundahl, L. H., Morrison, A., Dodge, N. C., . . . Jacobson, S. W. (2010). An event-related potential study of response inhibition in ADHD with and without prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, 34, 617-627. doi: 10.1111/j.1530-0277.2009.01130.x
- Chambers, C. D., Garavan, H., & Bellgrove, M. A. (2009). Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. *Neuroscience & Biobehavioral Reviews*, 33, 631-646. doi: 10.1016/j.neubiorev.2008.08.016
- Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences*. 2nd edn. Hillsdale, New Jersey: L Erlbaum.
- Conley, T. B. (2001). Construct validity of the MAST and AUDIT with multiple offender drunk drivers. *Journal of Substance Abuse Treatment*, 20, 287-295. doi: 10.1016/S0740-5472(01)00159-3

- Cornelius, B. L., Groothoff, J. W., van der Klink, J. J., & Brouwer, S. (2013). The performance of the K10, K6 and GHQ-12 to screen for present state DSM-IV disorders among disability claimants. *BMC Public Health*, *13*, 1-8.
doi:10.1186/1471-2458-13-128
- de Wit, S., & Dickinson, A. (2009). Associative theories of goal-directed behaviour: a case for animal–human translational models. *Psychological Research*, *73*, 463-476. doi:10.1007/s00426-009-0230-6
- de Wit, H., Crean, J., & Richards, J. B. (2000). Effects of d-amphetamine and ethanol on a measure of behavioral inhibition in humans. *Behavioral Neuroscience*, *114*, 830. doi:10.1037/0735-7044.114.4.830
- Dearden, J., & Payne, J. (2009). Alcohol and homicide in Australia. *Trends & Issues in Crime and Criminal Justice*, *372*, 1-6.
www.questia.com/library/p436748/trends-issues-in-crime-and-criminal-justice
- Denson, T. F., Aviles, F. E., Pollock, V. E., Earleywine, M., Vasquez, E. A., & Miller, N. (2008). The effects of alcohol and the salience of aggressive cues on triggered displaced aggression. *Aggressive Behavior*, *34*, 25-33.
doi:10.1002/ab.20177
- Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, *64*, 135.
doi: 10.1146/annurev-psych-113011-143750
- Dimoska-Di Marco, A., McDonald, S., Kelly, M., Tate, R., & Johnstone, S. (2011). A meta-analysis of response inhibition and Stroop interference control deficits in adults with traumatic brain injury (TBI). *Journal of Clinical and*

Experimental Neuropsychology, 33, 471-485.

doi:10.1080/13803395.2010.533158

Dolder, P. C., Holze, F., Liakoni, E., Harder, S., Schmid, Y., & Liechti, M. E. (2016).

Alcohol acutely enhances decoding of positive emotions and emotional concern for positive stimuli and facilitates the viewing of sexual images.

Psychopharmacology, 1-11. doi: 10.1007/s00213-016-4431-6

Dry, M. J., Burns, N. R., Nettelbeck, T., Farquharson, A. L., & White, J. M. (2012).

Dose-related effects of alcohol on cognitive functioning. *PLoS One*, 7, e50977. doi:10.1371/journal.pone.0050977

Dubois, B., Slachevsky, A., Litvan, I., & Pillon, B. (2000). The FAB A frontal

assessment battery at bedside. *Neurology*, 55, 1621-1626. doi:10.1212/WNL.55.11.1621

Earleywine, M., & Erblich, J. (1996). A confirmed factor structure for the Biphasic

Alcohol Effects Scale. *Experimental and Clinical Psychopharmacology*, 4, 107. doi:10.1037/1064-1297.4.1.107

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the

identification of a target letter in a nonsearch task. *Perception & Psychophysics*, 16, 143-149. doi:10.3758/BF03203267

Fals-Stewart, W., O'Farrell, T. J., Freitas, T. T., McFarlin, S. K., & Rutigliano, P.

(2000). The timeline followback reports of psychoactive substance use by drug-abusing patients: psychometric properties. *Journal of Consulting and Clinical Psychology*, 68, 134. doi:10.1037/0022-006X.68.1.134

Fassaert, T., De Wit, M., Tuinebreijer, W., Wouters, H., Verhoeff, A., Beekman, A.,

& Dekker, J. (2009). Psychometric properties of an interviewer-administered version of the Kessler Psychological Distress scale (K10) among Dutch,

- Moroccan and Turkish respondents. *International Journal of Methods in Psychiatric Research*, 18, 159-168. doi:10.1002/mpr.288
- Fillmore, M. T., Rush, C. R., & Hays, L. (2006). Acute effects of cocaine in two models of inhibitory control: implications of non-linear dose effects. *Addiction*, 101, 1323-1332. doi:10.1111/j.1360-0443.2006.01522.x
- Fillmore, M. T., & Vogel-Sprott, M. (2000). Response inhibition under alcohol: effects of cognitive and motivational conflict. *Journal of Studies on Alcohol*, 61, 239-246. doi:10.15288/jsa.2000.61.239
- Fillmore, M. T., Vogel-Sprott, M., & Gavrilesco, D. (1999). Alcohol effects on intentional behavior: dissociating controlled and automatic influences. *Experimental and Clinical Psychopharmacology*, 7, 372. doi:10.1037/1064-1297.7.4.372
- Fillmore, M. T., & Weafer, J. (2004). Alcohol impairment of behavior in men and women. *Addiction*, 99, 1237-1246. doi:10.1111/j.1360-0443.2004.00805.x
- Freeman, N., Friedman, R. S., Bartholow, B. D., & Wulfert, E. (2010). Effects of alcohol priming on social disinhibition. *Experimental and Clinical Psychopharmacology*, 18, 135-144. doi:10.1037/a0018871
- Gable, P. A., Mechin, N. C., & Neal, L. B. (2016). Booze cues and attentional narrowing: Neural correlates of virtual alcohol myopia. *Psychology of Addictive Behaviors*, 30, 377-382. doi:10.1037/adb0000130
- Giancola, P. R. (2007). The underlying role of aggressivity in the relation between executive functioning and alcohol consumption. *Addictive Behaviors*, 32, 765-783. doi:10.1016/j.addbeh.2006.06.015
- Giancola, P. R., Josephs, R. A., Parrott, D. J., & Duke, A. A. (2010). Alcohol Myopia Revisited: Clarifying Aggression and Other Acts of Disinhibition Through a

Distorted Lens. *Perspect Psychol Sci*, 5, 265-278.

doi:10.1177/1745691610369467

Goghari, V. M., & MacDonald, A. W. (2009). The neural basis of cognitive control:

Response selection and inhibition. *Brain and Cognition*, 71, 72-83.

doi:10.1016/j.bandc.2009.04.004

Grace, J., Moore, D., & Northcote, J. (2009). *Alcohol, risk and harm reduction:*

Drinking amongst young adults in recreational settings in Perth: National Drug Research Institute Perth.

Grant, N. K., & Macdonald, T. K. (2005). Can alcohol lead to inhibition or

disinhibition? Applying alcohol myopia to animal experimentation. *Alcohol Alcoholism*, 40, 373-378. doi:10.1093/alcalc/agh177

Heath, J., & Hardy-Vallée, B. (2015). Why do people behave immorally when drunk? *Philosophical Explorations*, 18, 310-329.

doi:10.1080/13869795.2015.996585

Hull, J. G., & Slone, L. B. (2004). Alcohol and self-regulation. In R. F. Baumeister &

K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 466–491). New York: Guilford.

Kamboj, S. K., Joye, A., Bisby, J. A., Das, R. K., Platt, B., & Curran, H. V. (2013).

Processing of facial affect in social drinkers: a dose–response study of alcohol using dynamic emotion expressions. *Psychopharmacology*, 227, 31-39.

doi:10.1007/s00213-012-2940-5

Kareken, D. A., Dziedzic, M., Wetherill, L., Eiler, W., 2nd, Oberlin, B. G.,

Harezlak, J., . . . O'Connor, S. J. (2013). Family history of alcoholism interacts with alcohol to affect brain regions involved in behavioral inhibition.

Psychopharmacology, 228, 335-345. doi:10.1007/s00213-013-3038-4

- Kessler, R. C., Andrews, G., Colpe, L. J., Hiripi, E., Mroczek, D. K., Normand, S.-L., . . . Zaslavsky, A. M. (2002). Short screening scales to monitor population prevalences and trends in non-specific psychological distress. *Psychological Medicine*, 32, 959-976. doi:10.1017/s0033291702006074
- Lithari, C., Klados, M. A., Pappas, C., Albani, M., Kapoukranidou, D., Kovatsi, L., . . . Papadelis, C. L. (2012). Alcohol affects the brain's resting-state network in social drinkers. *PLoS One*, 7, e48641. doi:10.1371/journal.pone.0048641
- Lopez-Caneda, E., Rodriguez Holguin, S., Cadaveira, F., Corral, M., & Doallo, S. (2014). Impact of alcohol use on inhibitory control (and vice versa) during adolescence and young adulthood: a review. *Alcohol and Alcoholism*, 49, 173-181. doi:10.1093/alcalc/agt168
- Lyvers, M., & Tobias-Webb, J. (2010). Effects of acute alcohol consumption on executive cognitive functioning in naturalistic settings. *Addictive Behaviors*, 35, 1021-1028.
- Manning, M., Smith, C., & Mazerolle, P. (2013). The societal costs of alcohol misuse in Australia. *Trends and Issues in Crime and Criminal Justice*, 454, 1-6.
- Marczinski, C. A., Abrams, B. D., Van Selst, M., & Fillmore, M. T. (2005). Alcohol-induced impairment of behavioral control: differential effects on engaging vs. disengaging responses. *Psychopharmacology*, 182, 452-459. doi:10.1007/s00213-005-0116-2
- Marczinski, C. A., & Fillmore, M. T. (2003). Preresponse cues reduce the impairing effects of alcohol on the execution and suppression of responses. *Experimental and Clinical Psychopharmacology*, 11, 110. doi:10.1037/1064-1297.11.1.110

- Marczinski, C. A., & Fillmore, M. T. (2005). Compensating for alcohol-induced impairment of control: effects on inhibition and activation of behavior. *Psychopharmacology*, *181*, 337-346. doi:10.1007/s00213-005-2269-4
- Martin, C. S., Earleywine, M., Musty, R. E., Perrine, M., & Swift, R. M. (1993). Development and validation of the biphasic alcohol effects scale. *Alcoholism: Clinical and Experimental Research*, *17*, 140-146. doi:10.1111/j.1530-0277.1993.tb00739.x
- Maudens, K. E., Patteet, L., van Nuijs, A. L., Van Broekhoven, C., Covaci, A., & Neels, H. (2014). The influence of the body mass index (BMI) on the volume of distribution of ethanol. *Forensic Science International*, *243*, 74-78. doi:10.1016/j.forsciint.2014.04.036
- McDonald, S., Gowland, A., Randall, R., Fisher, A., Osborne-Crowley, K., & Honan, C. (2014, May 12). Cognitive Factors Underpinning Poor Expressive Communication Skills After Traumatic Brain Injury: Theory of Mind or Executive Function? *Neuropsychology*, *28*, 801-811. doi:10.1037/neu0000089
- Meneses-Gaya, C., Zuardi, A. W., Loureiro, S. R., Hallak, J. E., Trzesniak, C., de Azevedo Marques, J. M., . . . Crippa, J. A. (2010). Is the full version of the AUDIT really necessary? Study of the validity and internal construct of its abbreviated versions. *Alcoholism: Clinical and Experimental Research*, *34*, 1417-1424. doi:10.1111/j.1530-0277.2010.01225.x
- Mitchell, I. J., Beck, S. R., Boyal, A., & Edwards, V. R. (2011). Theory of mind deficits following acute alcohol intoxication. *European Addiction Research*, *17*, 164-168. doi:10.1159/000324871
- Monahan, J. L., & Lannutti, P. J. (2000). Alcohol as social lubricant. *Human Communication Research*, *26*, 175-202.

- Montgomery, C., Ashmore, K. V., & Jansari, A. (2011). The effects of a modest dose of alcohol on executive functioning and prospective memory. *Human Psychopharmacology: Clinical and Experimental*, 26, 208-215.
- Morgan, A., & McAtamney, A. (2009). *Key issues in alcohol-related violence*: Canberra: Australian Institute of Criminology.
- Mulvihill, L., Skilling, T., & Vogel-Sprott, M. (1997). Alcohol and the ability to inhibit behavior in men and women. *Journal of Studies on Alcohol*, 58, 600-605.
- Osborne-Crowley, K., McDonald, S., & Rushby, J. A. (2016). Role of Reversal Learning Impairment in Social Disinhibition following Severe Traumatic Brain Injury. *Journal of the International Neuropsychological Society*, 22, 303-313. doi:10.1017/S1355617715001277
- Ostling, E. W., & Fillmore, M. T. (2010). Tolerance to the impairing effects of alcohol on the inhibition and activation of behavior. *Psychopharmacology*, 212, 465-473. doi:10.1007/s00213-010-1972-y
- Padula, C. B., Simmons, A. N., Matthews, S. C., Robinson, S. K., Tapert, S. F., Schuckit, M. A., & Paulus, M. P. (2011). Alcohol attenuates activation in the bilateral anterior insula during an emotional processing task: a pilot study. *Alcohol and Alcoholism*, 46, 547-552.
- Peacock, A., Bruno, R., Martin, F. H., & Carr, A. (2013). The impact of alcohol and energy drink consumption on intoxication and risk-taking behavior. *Alcoholism Clinical & Experimental Research*, 37, 1234-1242. doi:10.1111/acer.12086
- Pedersen, W. C., Vasquez, E. A., Bartholow, B. D., Grosvenor, M., & Truong, A. (2014). Are You Insulting Me? Exposure to Alcohol Primes Increases

- Aggression Following Ambiguous Provocation. *Personality & Social Psychology Bulletin*, 40, 1037-1049. doi:10.1177/0146167214534993
- Rueger, S. Y., & King, A. C. (2013). Validation of the Brief Biphasic Alcohol Effects Scale (B-BAES). *Alcoholism: Clinical and Experimental Research*, 37, 470-476. doi:10.1111/j.1530-0277.2012.01941.x
- Sayette, M. A., Creswell, K. G., Dimoff, J. D., Fairbairn, C. E., Cohn, J. F., Heckman, B. W., . . . Moreland, R. L. (2012). Alcohol and group formation a multimodal investigation of the effects of alcohol on emotion and social bonding. *Psychological Science*, 23, 869–878. doi:10.1177/0956797611435134.
- Sobell, L. C., & Sobell, M. B. (1992). Timeline follow-back. In R. Litten & J. Allen (Eds.), *Measuring alcohol consumption* (pp. 41 – 72). Totowa, NJ : Humana Press
- Sripada, C. S., Angstadt, M., McNamara, P., King, A. C., & Phan, K. L. (2011). Effects of alcohol on brain responses to social signals of threat in humans. *Neuroimage*, 55, 371-380. doi:10.1016/j.neuroimage.2010.11.062
- Steele, C. M., & Josephs, R. A. (1990). Alcohol myopia: Its prized and dangerous effects. *American Psychologist*, 45, 921.
- Steering Committee for the Review of Commonwealth/State Service Provision (SCRCSP) 2009. *Report on Government Services 2009*. Melbourne: Productivity Commission. Retrieved from <http://www.pc.gov.au/gsp/reports/rogs/2009>
- Stone, V. E., Baron-Cohen, S., & Knight, R. T. (1998). Frontal lobe contributions to theory of mind. *Journal of cognitive neuroscience*, 10, 640-656. doi:10.1162/089892998562942

- Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). Needham Height, MA: Allyn & Bacon.
- Vengeliene, V., Bilbao, A., Molander, A., & Spanagel, R. (2008). Neuropharmacology of alcohol addiction. *British Journal of Pharmacology*, 154, 299-315. doi:10.1038/bjp.2008.30
- von Hippel, W., & Gonsalkorale, K. (2005). "That is bloody revolting!" Inhibitory control of thoughts better left unsaid. *Psychological Science*, 16, 497-500.
- Wager, T. D., Sylvester, C.-Y. C., Lacey, S. C., Nee, D. E., Franklin, M., & Jonides, J. (2005). Common and unique components of response inhibition revealed by fMRI. *Neuroimage*, 27, 323-340. doi:10.1016/j.neuroimage.2005.01.054
- Weafer, J., & Fillmore, M. T. (2008). Individual differences in acute alcohol impairment of inhibitory control predict ad libitum alcohol consumption. *Psychopharmacology*, 201, 315-324. doi:10.1007/s00213-008-1284-7
- Weafer, J., & Fillmore, M. T. (2012). Comparison of alcohol impairment of behavioral and attentional inhibition. *Drug and Alcohol Dependence*, 126, 176-182. doi:10.1016/j.drugalcdep.2012.05.010
- Weafer, J., & Fillmore, M. T. (2016). Low-Dose Alcohol Effects on Measures of Inhibitory Control, Delay Discounting, and Risk-Taking. *Current Addiction Reports*, 3, 75-84. doi:10.1007/s40429-016-0086-y
- Wennberg, P., & Bohman, M. (1998). The timeline follow back technique: Psychometric properties of a 28-day timeline for measuring alcohol consumption. *German Journal of Psychiatry*, 2, 62-68.
- Retrieved from: <http://wwwuser.gwdg.de/~bbandel/gjp-article-wennberg.htm>

Appendix A Ethics Approval

Office of Research Services
University of Tasmania
Private Bag 1
Hobart Tasmania 7001
Telephone + 61 3 6296 7479
Facsimile + 61 3 6296 7145
Email HumanEthics@utas.edu.au
www.research.utas.edu.au/human_ethics/

HUMAN
RESEARCH
ETHICS
COMMITTEE
(TASMANIA)
NETWORK



17 May 2016

Dr Cynthia Honan
C/o- Psychology

Sent via email

Dear Dr Honan

REF NO: H0015633

TITLE: Alcohol Intoxication and social cognition: an examination of
perception and response to social information

Document
Application Form – NEAF
Protocol – Alcohol Study
Psychology Peer Review

The Tasmanian Health and Medical Human Research Ethics Committee considered and approved the above documentation on 10 May 2016 to be conducted at the following site(s):

University of Tasmania

Please ensure that all investigators involved with this project have cited the approved versions of the documents listed within this letter and use only these versions in conducting this research project.

This approval constitutes ethical clearance by the Health and Medical HREC. The decision and authority to commence the associated research may be dependent on factors beyond the remit of the ethics review process. For example, your research may need ethics clearance from other organisations or review by your research governance coordinator or Head of Department. It is your responsibility to find out if the approvals of other bodies or authorities are required. It is recommended that the proposed research should not commence until you have satisfied these requirements.

All committees operating under the Human Research Ethics Committee (Tasmania) Network are registered and required to comply with the *National Statement on the Ethical Conduct in Human Research* (NHMRC 2007 updated 2014).

Therefore, the Chief Investigator's responsibility is to ensure that:

- (1) The individual researcher's protocol complies with the HREC approved

protocol.

(2) Modifications to the protocol do not proceed until approval is obtained in writing from the HREC. Please note that all requests for changes to approved documents must include a version number and date when submitted for review by the HREC.

(3) Section 5.5.3 of the National Statement states:

Researchers have a significant responsibility in monitoring approved research as they are in the best position to observe any adverse events or unexpected outcomes. They should report such events or outcomes promptly to the relevant institution/s and ethical review body/ies and take prompt steps to deal with any unexpected risks.

The appropriate forms for reporting such events in relation to clinical and non-clinical trials and innovations can be located at the website below. All adverse events must be reported regardless of whether or not the event, in your opinion, is a direct effect of the therapeutic goods being tested. <http://www.utas.edu.au/research-admin/research-integrity-and-ethics-unit/new/human-ethics/human-research-ethics-review-process/health-and-medical-hrec/managing-your-approved-project>

(4) All research participants must be provided with the current Patient Information Sheet and Consent Form, unless otherwise approved by the Committee.

(5) The Committee is notified if any investigators are added to, or cease involvement with, the project.

(6) This study has approval for four years contingent upon annual review. A Progress Report is to be provided on the anniversary date of your approval. Your first report is due 10 May 2017. You will be sent a courtesy reminder closer to this due date.

(7) A Final Report and a copy of the published material, either in full or abstract, must be provided at the end of the project.

Should you have any queries please do not hesitate to contact me on (03) 6226 2764.

Yours sincerely

Heather Vall
Ethics Administrator
Office of Research Services
Email: Heather.vall@utas.edu.au
University of Tasmania
Private Bag 01 Hobart Tas 7001

Appendix C Information Sheet



School of Psychology

University of Tasmania

Information Sheet

The Impact of Alcohol Consumption on Social Ability

April 2016

Introduction

You are invited to participate in an experiment examining the effect of alcohol on social ability. The research is being conducted by Miss Emma Johnson and Miss Sarah Skromanis in partial fulfilment of the requirements of an Honours degree at the University of Tasmania. Emma and Sarah are being supervised by Dr Cynthia Honan, a Clinical Neuropsychologist and Lecturer from the Discipline of Psychology, School of Medicine, University of Tasmania. The researchers can be contacted as following: Emma Johnson (emma.johnson@utas.edu.au; Ph: 03 6324 3266); Sarah Skromanis (sarah.skromanis@utas.edu.au; Ph: [03 6324 3266](tel:0363243266)); Dr Cynthia Honan (cynthia.honan@utas.edu.au; Ph: 03 6324 3266).

What is the purpose of the study?

The purpose of this study is to investigate whether alcohol interferes with social ability. Emotion perception and theory of mind ability (ability to understand the thoughts and behaviours of others), and the ability to inhibit automatic social responding will be specifically examined. These abilities will be assessed using cognitive tasks.

Who can participate?

We are currently seeking participants who are:

- Aged 18-35 years
- Speak and read fluent English
- Completed Year 10 or equivalent
- Normal or corrected-to-normal vision
- Healthy (no history of significant neurological disorder or current psychiatric disorder, significant intellectual disorder, alcohol/drug dependence, regular tobacco use, or chronic health problems)
- Regular alcohol consumers (minimum consumption of 2 standard alcoholic drinks on one occasion in the preceding month)
- Not currently using illicit drugs (i.e. use in the past six months)

- Able to attend the Newnham campus of the University of Tasmania for up to 3 hours between 9am and 7pm (session lengths are an estimate only).

What does participation in the study involve?

This research will be conducted in Buildings O and N at the Newnham Campus, University of Tasmania. Interested individuals will complete some online screening questionnaires that will ask for your demographic details (e.g., age, sex, education), height and weight (to calculate Body Mass Index), medical history, psychological functioning, and use of alcohol. Eligible participants will be contacted to attend the Newnham campus for an experimental session conducted between 9am and 7pm.

Experimental sessions:

At the beginning of the session participants will consume a 150ml beverage before completing questionnaires asking about alcohol intake in the previous month and current mood, and brief cognitive tasks assessing basic emotion perception and inhibition ability. Participants will then be asked to consume a 750ml beverage that will contain either a placebo or alcohol. Alcohol administered will be a maximum of 6 standard alcoholic drinks. Participants will not be informed of the beverage content administered in each session until the conclusion of the session.

After consuming the beverage, participants will be asked to complete one emotion recognition task, two computerised laboratory tasks assessing motor responses and inhibition ability, and one social disinhibition task. A breathalyser will be used to monitor participants' breath alcohol concentration throughout the duration of the study. Throughout testing, participants will also be asked to complete several scales assessing their feeling of intoxication and impairment.

While it is estimated that the experimental tasks will take approximately 100 minutes to complete, some participants may be required to remain in the laboratory for a total of 3 hours to ensure each participant records two consecutive breath alcohol readings of .03% or less (.00% for Provisional licence holders intending to drive). These times are an estimate only as individual rates of alcohol absorption and elimination may vary. Participants will be debriefed regarding the order of dose administration at the conclusion the session.

What are the restrictions regarding participating?

Participants will be asked to fast from food for 4 hours prior to each experimental session and abstain from caffeine for 8 hours and alcohol and over-the-counter medication for 24 hours prior to each session. Participants will be asked to abstain from illicit drugs and tobacco for the duration of participation. Participants will be asked to consume two slices of toast with their choice of spread one hour prior to each session.

At the end of each session, participants will remain at leisure (with food and entertainment provided) until they attain two consecutive breathalyser recordings of 0.03% or less measured 15 minutes apart. Participants holding their provisional driver licence, who are intending to drive will be required to remain in the laboratory

until two consecutive BrAC measurements are recorded at .00%. Participants holding their provisional licence who are not intending to drive, will be able to leave the laboratory at .03% BrAC if they sign a declaration in which they agree to be escorted by a nominated guardian to their place of residence and accompanied for a two hour period following session completion. The nominated guardian must be an adult aged 18 years or older who: (i) holds their provisional or full driver licence (ii) directly collects the participant from the research premises and meets the researcher in-person, and (iii) signs a declaration agreeing to escort the participant directly to their place of residence and accompany the participant for the two hour period following session completion. The researcher reserves the right to retain participants in the laboratory until .03% BrAC for those holding their full driver licence and .00% BrAC for those holding their provisional licence when it is deemed unsafe for the participant to leave at .03% BrAC.

What are the benefits of participating?

Your participation will help us enhance our knowledge of the effects of alcohol on social ability, and specifically, the mechanisms underlying social disinhibition, theory of mind and emotion perception. This knowledge can be used to educate people regarding the potential outcomes of alcohol intoxication on social functioning and will inform further research that aims to investigate alcohol related social difficulties.

What are the risks associated with participating?

There are no anticipated risks of this research. However, if in the unlikely event you experience negative side-effects, please inform the experimenter and the necessary assistance will be sought and provided. We ask that participants refrain from consuming alcohol or operating heavy machinery for four hours post-session.

Is there any reimbursement for participation?

Students of the University of Tasmania who are undertaking KHA111/112 unit will receive up to three hours research participation credit for their time. This will depend on the time required to return to a .03% BrAC reading (or .00 BrAC for provisional drivers) as specified above. Participants who are not undertaking KHA111/112 units will receive a Village Cinemas movie ticket as recompense for their time. Participants who do not complete the full schedule of sessions will not receive a movie ticket, unless withdrawal is necessary due to an unexpected adverse physiological reaction to the investigatory products.

How do I volunteer to participate? What if I want to withdraw from participating? Participation in this study is voluntary. By signing the attached consent form, you are indicating that you are aware of the nature of the study and wish to participate. While we would be pleased to have you participate, we respect your right to decline. There will be no consequences to you if you decide not to participate. If you decide to discontinue participation at any time, you may do so without providing an explanation. However, you will be required to remain in the laboratory until your breath alcohol concentration measurement equals 0.03% or less on two separate occasions measured 15 minutes apart.

What will happen to the information I provide?

All information collected will be kept confidential. Each participant will be assigned a treatment code and individual participant data will be identifiable only by that code. All of the data will be stored on password protected secure computers or in a locked cabinet in the Discipline of Psychology, School of Medicine for a minimum of five years after the publication of any academic journal articles, at which point all questionnaires will be destroyed using a paper shredder and electronic data will be deleted. The screening questionnaire will be securely destroyed immediately on completion of the study and that any information provided by the participant on the questionnaire will be identifiable only by participant number, kept confidential, and viewed only by the experimenter.

Who do I contact if I have any queries?

If you would like to discuss any aspect of this study please contact Emma Johnson (emma.johnson@utas.edu.au) or Sarah Skromanis (sarah.skromanis@utas.edu.au). Alternatively, you can contact Dr Cynthia Honan on (03) 6324 3266 or email cynthia.honan@utas.edu.au.

How do I find out the results of the study?

A summary of the results will be available on the Research webpage of the Discipline of Psychology, University of Tasmania (<http://www.utas.edu.au/health/study/psychology>). Results of the study can also be provided by contacting the researchers directly.

Who do I contact if I have a complaint about the study?

This study has been approved by the Tasmanian Health and Medical Human Research Ethics Committee. If you have concerns or complaints about the conduct of this study should contact the Executive Officer of the HREC (Tasmania) Network on (03) 6226 7479 or email human.ethics@utas.edu.au. The Executive Officer is the person nominated to receive complaints from research participants. You will need to quote .

Who do I contact if I wish to speak to someone about my alcohol or drug use, or mental health?

As aforementioned, a number of simple screening questionnaires will be administered assessing psychological functioning and alcohol and other drug use. Whilst it is not anticipated that these questionnaires will cause distress, please do not hesitate to let the researcher know if you do not wish to fill them in. If you are concerned about your drinking or mental health, please contact the Tasmanian Alcohol Drug Information Service 1800 811 994 or Lifeline 13 11 14 (both services available 24 hours a day).

**Thank you for taking the time to consider this study.
If you wish to take part in it, please sign the attached consent form.
This information sheet is for you to keep.**

Appendix D Consent Form



School of Psychology
University of Tasmania

Consent Form

The Impact of Alcohol Consumption on Social Ability

1. I have read and understood the 'Information Sheet' for this project.
2. The nature and possible effects of the study have been explained to me.
3. I understand that because of my prior participation in eligibility screening session in which I have completed measures of psychological distress and alcohol use, as well as reporting my correct demographic data (age, sex, height and weight) that I am eligible to participate in the study.
4. I understand that I will be asked to abstain from food for 4 hours, caffeine-containing products for 8 hours, and alcohol and prescription medication for 24 hours prior to each session, and illicit drugs and tobacco for the duration of the study. I will be asked to consume a standard meal 60 minutes prior to the experimental session.
5. I will be asked to sign a declaration and complete a breath alcohol concentration measurement (via a breathalyser) to confirm my abstinence at the start of each session.
6. I understand that in the experimental session I may be given a maximum of 6 standard alcoholic drinks, and that I will not be informed of the specific contents of the beverage until the conclusion of testing. I understand that after beverage consumption, I will be asked to complete a number of computerised laboratory behavioural performance tasks during which my behavioural responses will be recorded. I understand that my breath alcohol concentration (as measured via a breathalyser) will be recorded throughout the session, and that I will be asked about my perception of my intoxication and level of impairment.
7. I understand that the study involves attending the Newnham campus of the University of Tasmania (Buildings O and N) for one 100 minute experimental session.
8. I understand that I will be asked to remain in the laboratory until my blood alcohol concentration equals 0.03% or less on two occasions measured 15 minutes apart. This may mean remaining in the laboratory for approximately 3 hours in total.
9. I acknowledge that I have been advised to refrain from drinking alcohol or operating a vehicle or other heavy machinery for four hours after the end of the experimental session.

10. I understand that if I hold a provisional driver licence and I intend to drive I will be required to remain in the laboratory until my breath alcohol concentration is .00% on two consecutive occasions. I understand that if I hold a provisional driver licence and do not intend to drive I will be able to leave the laboratory at .030% BrAC after signing a declaration in which I agree to be escorted by my nominated legal adult to my place of residence and be accompanied for a two hour period following session completion. I understand that the nominated legal guardian must be an adult aged 18 years or older who: (i) holds their provisional or full driver licence (ii) directly collects me from the research premises and meets the researcher in-person, and (iii) signs a declaration agreeing to escort me directly to my place of residence and accompany me for a two hour period following session completion. Furthermore, I understand that the researcher reserves the right to retain participants in the laboratory until .03% BrAC for those holding their full driver licence and .00% BrAC for those holding their provisional licence when it is deemed unsafe for the participant to leave at .03% BrAC. I acknowledge that I have been advised to refrain from drinking alcohol or operating a vehicle or other heavy machinery for four hours after the end of experimental sessions.

11. I understand that I will be entered into a draw to win one of five double movie ticket passes for my participation in this study. I understand that if I am a KHA111/112 student I can opt to be reimbursed up to three hours research participation credit in addition instead of entering the prize draw. If I withdraw from the study prior to concluding all sessions I will not be eligible for reimbursement, unless the withdrawal is due to an unexpected adverse event occurring as a consequence of ingesting the beverage.

12. I understand that, while there are no anticipated risks associated with this study, I should inform the experimenter immediately if any unexpected negative side-effects are experienced. I understand the experimenter will immediately cease the session and seek the necessary assistance.

13. I understand that the researchers will maintain my confidentiality and that any information I supply to the researcher(s) will be used only for the purposes of the research. My data will only be identifiable by an individual numerical participant code.

14. I understand that the screening questionnaire will be securely destroyed immediately on completion of the study and that any information I provide on the questionnaire will be identifiable only by my participant number, kept confidential, and viewed only by the experimenter.

15. I understand that all research data will be securely stored on the University of Tasmania premises for at least five years, and will then be securely destroyed when no longer required.

16. I agree that research data gathered from me for the study may be published provided that I cannot be identified as a participant.

17. I agree to participate in this investigation and understand that I may withdraw at any time without any effect, and if I so wish, may request that any data I have supplied to date be withdrawn from the research.

18. Any questions that I have asked have been answered to my satisfaction.

Name of Participant:

Signature:

Date:

Statement by Investigator

☐ I have explained the project & the implications of participation in it to this volunteer and I believe that the consent is informed and that he/she understands the implications of participation

If the Investigator has not had an opportunity to talk to participants prior to them participating, the following must be ticked.

☐ The participant has received the Information Sheet where my details have been provided so participants have the opportunity to contact me prior to consenting to participate in this project.

Name of Investigator:

Signature:

Date:

Appendix E Widmark Equation

$$\text{Alcohol Dose (mg)} = W\rho(C_1 + \beta t)$$

W Participants body weight (kg),

ρ Distribution of alcohol in the body

C_1 target breath alcohol concentration (BrAC; g/100mL),

t Time (Hours)

β Rate of alcohol elimination. Set at 0.015g/100mL/hour.

Note: Final alcohol dose (mg) is divided by 0.8 to achieve a dose in millilitres.

Appendix F Social Disinhibition Task Sample Items

Sample theory of mind item:

Linda was thanking Angus for the great game. Angus' wife was waiting patiently for them to finish talking.

Tell me what you think of Linda, go ahead.



Sample non-theory of mind item:

Nadine was annoyed that her boyfriend Jack forgot her birthday.

Tell me what you think of Jack go ahead.

